INTRODUCTION

Thanks to the introduction of potent regimens of combined antiretroviral therapy (cART), since mid-1996 the frequency of opportunistic infections had a dramatic decline among HIV-infected patients, and the life expectancy of this patient population significantly increased. At the same time, due to the increased mean age of HIV-infected population, and a series of potential supporting factors (i.e. persisting immune system imbalance, exposure to drugs and carcinogenic substances, continued latency of potentially oncogenic viruses, HIV infection itself), haematological and especially solid organ malignancies showed a proportional increase during time. As a consequence, besides the traditionally reknown AIDS-related cancers (i.e. Kaposi’s sarcoma, non-Hodgkin’s lymphoma, and primary central nervous system lymphoma, together with cervical cancer added to the list since the year 1993), there is a worldwide emergence of broad-spectrum haematological and especially solid organ neoplasms, which also seem to occur at a younger age, and encompass a greater aggressivity and a more frequent and more rapid mortality compared with the general population [1-3]. The possible occurrence of multiple (concurrent or subsequent) malignancies, has been also reported during recent years, after the availability of cART [1-5]. The differential diagnosis and therefore the treatment of these disorders are usually delayed and hampered by co-existing disorders, especially chronic-relapsing infectious complications, related or unrelated to the eventual, underlying HIV-associated immunodeficiency [1-5].

In particular, among neoplastic complications of the ear, nose, and throat (ENT) districts, those affecting the nose and the rhinopharynx remain somewhat rare among HIV-infected patients, with extranodal lymphomas burdened by a series of possible local complications, prevailing over squamous adenocarcinoma and other solid tumours, differently from the disease distribution found in the general population [6-8].

The diagnosis of tumour-like rhinopharyngeal lesions remains difficult when an expected lymphocyte-monocyte hyperplasia involving the local lymphoid districts occurs in HIV-infected subjects, who are prone to show a reactive lymphoid proliferation descending from HIV disease itself, and eventual concurrent infections, like that caused by Epstein-Barr virus [6-8].

Moreover, slowly progressive infections possibly caused by a large spectrum of pathogenic microorganisms (especially atypical mycobacteria, and Mycobacterium tuberculosis), may occur as space-occupying masses of the nose.
and/or the rhinopharynx [9]. They tend to mimick other local complications, including neoplastic ones, and remarkably complicate the differential diagnostic process of these lesions. Finally, similarly to the occurrence of tobacco smoking as a known risk factor of upper aerodigestive tract malignancies, also a prolonged exposure to recreational substances (including inhalation of drugs like cocaine and heroin), has been described as a possible supporting factor of rare, anecdotal cases of heterogeneous tumours of the nasal cavity among HIV-infected subjects, with or without local superinfections caused by the coexistence of ulcerative mucosal lesions, and a varied resident flora colonizing the upper respiratory tract [10-12]. With regard to the lesions of the nasal septum and nasal cartilage and sinusal bone tissue, when excluding congenital lesions, all acquired ones usually show aspecific imaging findings also at sophisticated study techniques, like X-ray films, computerized tomography (CT) scans, magnetic resonance imaging (MRI), and so on. These lesions may be caused by a very broad spectrum of causes, including nasal trauma (involving surgical interventions and invasive diagnostic procedures, accidents, but also rhinotillexomania), exposure to toxic substances (including local decongestants and inhaled cocaine or other drugs), inflammatory diseases (i.e. sarcoidosis, reparative granulomas, Wegener’s granulomatosis, and other collagen vascular diseases), multiple infection (caused by bacteria, mycobacteria, fungi, or associated microorganisms), or malignancies (whose large spectrum of disorders includes carcinomas, sarcoma, Pindborg tumor, angiomyxoma, hemangioma, neuroendocrine tumor, schwannoma, and primary or secondary lesions of lymphoproliferative disorders) [12].

Aim of our present report is to describe an extremely rare occurrence of associated rhinopharyngeal actinomycosis and squamous adenocarcinoma in a HIV-infected patients treated with cART and with some specific and local risk factors (cigarette smoking, long-term inhalatory substance abuse, and a half-professional mushroom-truffle search and evaluation by smell).

The histopathological and imaging diagnostic work-up of our patient’s disease, and its therapeutic and outcome features, are presented and discussed on the ground of the available literature evidences.

CASE REPORT

A 49-year-old male ex-i.v. drug user was diagnosed with HIV infection since 13 years, while a concurrent chronic HCV hepatitis was disclosed since 10 years. Due to a very low compliance to clinical and laboratory controls, both antiretroviral therapy and chemoprophylaxis against the most common HIV-associated opportunistic infections were denied by our patient during his first three years of follow-up, when active drug addiction was still present. Ten years ago, our patient was hospitalized owing to the development of an AIDS-defining illness (neurotoxoplasmosis). At that time, he experienced his nadir of absolute CD4+ lymphocyte count (36 cells/µL only).

After successful cure of this central nervous system opportunistic complication, our patient finally accepted a combined antiretroviral therapy (cART), initially conducted with didanosine, stavudine, and indinavir, together with a secondary prophylaxis for toxoplasmosis, carried out with cotrimoxazole, in order to prevent a pneumocystosis, too. Despite a limited adherence to antiretroviral therapy (not exceeding 70-80% of recommended doses, as assessed on the ground of spontaneous patient’s declarations, and monthly drug accountability carried out at our dedicated HIV outpatient service), a progressive recovery of the CD4+ count at levels above 200 cells/µL, allowed the discontinuation of cotrimoxazole prophylaxis eight months after hospital discharge. Even though a complete suppression of HIV viremia was reached after the first six months of cART, it was never sustained subsequently, probably due to the limited patient’s adherence to different, alternative regimens of cART proposed in order to enhance his adherence. During the subsequent laboratory follow-up, plasma HIV-RNA levels ranging from 450 and 4,300 copies/mL were disclosed, in absence of further occurrences of negative viremia.

During the further follow-up until recently, our patient progressively abandoned i.v. drug addiction but resorted to a “recreational”, inhalatory use of both cocaine and heroin. Moreover, he continued a personal semi-professional hobby regarding frequent mushroom and truffle search, and continued tobacco (cigarette) smoking, which lasted since the age of 16 years.

Despite low adherence levels to four different lines of cART, the stable and sufficiently satisfactory immune recovery, as established by a
CD4+ count always above 400 cells/µL (above 26-30% of total T-lymphocytes, with a peak value of 486 cells/µL in the year 2003), and the negligible clinical history, allowed our patient to maintain his low-adherence cART regimen, until a genotypic resistance testing of 12 months ago performed with rising plasma HIV-RNA levels (5,190 copies/mL). This last virological assay detected multiple nucleoside/nucleotide resistance mutations, complete resistance to both available non-nucleoside reverse transcriptase inhibitors, and multiple protease inhibitors resistance mutations, so that a cART regimen including lamivudine, abacavir, and fosamprenavir-ritonavir was introduced three months before the occurrence of the present disease, although patient’s adherence levels remained unsatisfactory.

Three months after the introduction of the last therapeutic line, when HIV-associated virological and immunological parameters showed a CD4+ lymphocyte count of 418 cells/µL, and a plasma viremia of 2,620 HIV-RNA copies/mL, our patient started to complain of severe subacute sinusitis-rhinitis-like signs and symptoms, including mucous-purulent discharge (predominantly from the left naris), poorly controlled by local and inhalatory therapy, and a systemic broad-spectrum antibiotic treatment, associated with non-steroidal anti-inflammatory drugs and mucolytics. After repeated ENT specialist consultations, our patient underwent a contrast-enhanced CT scan and MRI of the face and brain, which pointed out an aggressive form of rhinitis and sinusitis, with a severe inflammatory involvement of maxillary and ethmoidal sinuses (Figures 1 and 2).

Subsequently, a fiberoptic rhinoscopy (Figure 3) with associated multiple biopsies, culture, and histopathological examination, disclosed a predominantly necrotic tissue with abundant fibrin deposition, and actinomycosis-like “sulphur” granules surrounded by a relevant monocyte-macrophage cell infiltrate. Culture search for aerobe pathogens, fungi, and mycobacteria tested negative.

Based on this diagnosis of actinomycosis, a specific antimicrobial treatment with oral amoxicillin-clavulanate was started, and changed after seven days with i.v. imipenem (at 1 g, twice daily), for further 25 days, but local manifestations did not improve, as well as local tenderness and nasal discharge, so that a surgical approach to the lesion was established together with ENT consultants. The ENT surgical intervention included a left anterior ethmoidecto-

Figures 1 and 2 - Contrast-enhanced CT scan and gadolinium-enhanced magnetic resonance imaging (MRI). A solid mass with dishomogeneous enhancement, which occupies the whole nasal cavity, is demonstrated.
my, a left meatotomy, and a thorough revision of the left nasal fossa, where a solid neoformation was removed. The histopathologic examination of this surgical specimen surprisingly showed the concurrence of a poorly differentiated squamocellular carcinoma, with concurrent basaloid aspects, together with Actinomyces-like bacterial colonies. Although regional adenopathic involvement was excluded, after diagnosis a local, large, stage T3 neoplasm, a combined radiotherapy-chemotherapy regimen was started, after Oncology consultation. A local radiotherapy was started and continued for two months, together with a concurrent chemotherapy (i.v. cysplatinum at 100 mg/m² administered after the days 1, 22, and 43 of radiotherapy cycles).

The concomitant cART, although taken again with limited compliance, was changed into an association of tenofovir, emtricitabine, and atazanavir-ritonavir, which led to a negative HIV viremia after 5 weeks (HIV-RNA below 40 copies/mL), while our patient’s CD4+ count dropped and remained around 200-250 cells/µL. During the intensive ENT follow-up, a further fiberoptic endoscopy was performed two months after the first diagnosis of malignancy, and multiple ethmoidal biopsy studies demonstrated the persistence of an intense, subacute inflammatory process, without evidence of specific infection, and without evidence of residual local cancer lesions, at both microbiological and histopathological studies. One month later, a sudden deterioration of clinical and neurological conditions were interpreted as signs and symptoms of a severe central nervous system involvement, rapidly worsening into a deep comatous state, and an overwhelming cachexia, which led our patient to death in two weeks. A further contrast-enhanced TC scan obtained a two weeks before patient’s death demonstrated a diffuse nodular

Figure 3 - Endoscopic examination, carried out by a fiberoptic instrumentation. The left nasal fossa is completely occupied by a tissue of solid macroscopic appearance. The implant/cleavage basis cannot be defined.
lesions of the basal nuclei, markedly enhanced by contrast, some of them complicated by an evident surrounding oedema, interpreted as secondary spread of the primary rhino-sinusal carcinoma (Figure 4).

**DISCUSSION**

Actinomycosis is a rare human disease caused by slow-growing, anaerobic, filamentous, Gram-positive, catalase-negative organisms, which usually involves the cervical-facial region including the oral cavity, paranasal sinuses, parotid glands, orbit, neck, and anatomically close regions where the infection spreads from contiguous areas. Only a few reported episodes of actinomycosis involve the lower respiratory tract (the so-called thoracic form), and the gastrointestinal tract (the so-called abdominal form), after a primary focus usually localized in the upper airways. Hematogenous dissemination and distant foci of infection are extremely rare.

From a bacteriological point of view, although *Actinomyces israelii* is the most frequent microorganism responsible of human actinomycosis, other species (including *Actinomyces naeslundii, Actinomyces viscosus, Actinomyces odontolyticus*, and the close species *Arachnia propionica*), have been anecdotally reported in human disease [13, 14]. Historically mistaken with fungi because of their light microscopic aspect, and their difficulty to grow in regular mediums and aerobic conditions, *Actinomyces* spp bacteria are typically environmental in origin, since they represent saprophytic organisms usually found in the soil and dust, but they are also capable of colonizing the oral cavity, especially when poor oral hygiene and underlying dental and parodontal diseases are of concern, as in drug addicts. Sparse cases of cervical-facial actinomycosis have been attributed to nasal trauma or surgical manipulation, too [13, 14]. Actinomycosis is diagnosed with a positive culture (which actually is very difficult to be obtained in the current clinical practice, due to the fastidious culture requirements, and the need to rely on a very rapid transportation to a specialized laboratory, and an immediate incubation in microaerophilic or anaerobic environment), so that the diagnosis is more often obtained by detecting the typical colonies and “sulfur” granules in histopathologic specimens.

In its typical involvement of cervical-facial structures, the clinical appearance of actinomycosis may vary from enlarging, painless, firm or fluctuant swelling, either complicated or not by ulcerated lesions [11, 13, 14]. The late evolution towards multiple mucous-cutaneous fistulizations draining the typical “sulphur” granules is frequently a diagnostic clue. Radiological examination and other imaging techniques (CT and/or MRI) of the paranasal sinuses and the nose allow to exclude osteolytic processes (typical of a malignant process), and are very useful prior to the unavoidable resort to invasive diagnostic techniques, which is essentially based on multiple biopsy, and culture together with histopathology, and prior to the frequent surgical excision [12, 15]. In fact, save limited episodes, the treatment of choice is based on a surgical enucleation of the entire lesion with debridement of necrotic tissues, associated with a long-term antibiotic treatment, carried out with beta-lactam derivatives, which are effective against bacteria belonging to *Actinomyces* spp.

When considering actinomycosis and the ENT
districts, the slow, indolent progression of these atypical bacterial lesions pose unavoidable problems of differential diagnosis, also reflecting into a frequently delayed management. For example, four episodes of nasal-rhinopharyngeal actinomycosis just imitating nasopharyngeal carcinoma have been successfully cured after prolonged antibiotic therapy only (without resorting to surgery), as reported in 2000 by Chaing et al. [16]. Another anecdotal report of nasopharyngeal actinomycosis occurred in 2004 but surgical debridement became necessary to ensure cure, together with long-term antimicrobial treatment [17]. Moreover, actinomycosis mimicking a case of carcinoma of the maxillary sinus has been also described, as well as the case of a chronic sinus actinomycosis which may masquerade as headache or other subacute-chronic facial-brain disorders [18, 14]. More specifically, an actinomycosis of the internal nose, of the nasal septum, the nasal wall, and the nasal turbinates, remains an extremely rare occurrence, although an apparently novel species, named Actinomyces nasicola, has been recently isolated for the first time from a human nose, as a saprophytic organism of the local flora [11, 13, 14, 19-22]. In the majority of the sparsely reported cases, intranasal actinomycosis presented with the appearance of a local neoplasm [20]. Finally, an exceptional case of primary cutaneous involvement of the external surface of the nose has been recently described [23].

During the history of HIV pandemic, regardless of the availability of potent cART regimens, the prevalence of actinomycosis has remained low, despite the possible, even severe, impairment of cellular and humoral immunity that accompany HIV disease progression, so that actinomycosis has not been considered among opportunistic infections possibly associated with advanced HIV infection and AIDS [20]. Among patients with HIV disease and AIDS, very few cases of actinomycosis have been reported as anecdotal observations or small case series, which had a locally unfavourable outcome especially in the pre-cART era [20, 24-27]. Combined illnesses including actinomycosis plus other infectious and non-infectious (i.e. fungal) diseases have been occasionally reported, and the diagnostic and treatment difficulties have been repeatedly underlined [24, 25]. After the introduction of cART, heterogeneous disease localizations of actinomycosis have been exceptionally reported during the course of HIV infection, including an isolate case of esophageal actinomycosis complicated by multiple, non-healing organ ulcers, and an episode of pulmonary actinomycosis secondary to bacteremic dissemination [26, 27]. In only one case, a nasal actinomycotic mass was concurrent with secondary lesions from an underlying malignant disease (choriocarcinoma) in a patient with HIV infection, while no cases of associated actinomycosis plus a local, underlying squamous cell adenocarcinoma of the same ENT district have occurred until now in both HIV-infected and HIV-non-infected subjects, to the best of our knowledge [28].

From an epidemiological and pathogenetic point of view, the inhalation of recreational substances (cocaine and heroin), and the nasal exposure to dust and mould (as in our case), seems to predispose to the development of serious ENT pathologic processes, like septal defects and ulcerations, superinfections, squamous cell carcinomas but also local tumors other than adenocarcinoma, as well as a destruent midfacial osteomyelitis, which occurred in a chronic cocaine abuser [10, 11]. Intranasal nasal-septal lesions and ulcerations are a consequence of the well known cocaine’s vasoconstrictive properties, and the consequent decrease of oxygen tension of intranasal tissue may greatly facilitate the growth of anaerobic pathogens, including those belonging to the rare human pathogens belonging to Actinomyces spp. [11]. A non-HIV-infected i.v. drug abuser suffered from a rare, severe, and prolonged (six month-long) episode of skin and soft-tissue abscess caused by a concurrent Actinomyces odontolyticus and two Prevotella spp. organisms; a possible origin of this original mixed flora from the oral cavity was suspected, since the patient referred licking his needle prior to subcutaneous cocaine injection [29].

Coming to the distinctive features of our reported case, in our patient the first diagnosis of rhinopharyngeal actinomycosis was oriented also by relevant epidemiological and behavioural issues: the chronic cocaine and heroin inhalation was recognized as a relevant risk factor for intranasal ulcerative lesions which may support superinfections, and the patient’s preferred hobby of searching mushrooms and truffles became of real concern, due to the patient’s referred habit to smell and the repeated, unavoidable local contact and intranasal aspiration of mould and dust, which represent the known environmental reservoir of Actinomyces spp bacteria.
Like other fastidious, anaerobe organism requiring stringent conditions for microbiological culture and isolation, the diagnosis was posed on the characteristic appearance of granulomatous lesions (“sulfur” granules), at targeted intranasal biopsy and related histopathology studies. The underlying cancer lesions of the same region has not been initially suspected, since the first fiberoptic rhinoscopy and biopsy did not allow the recognition of malignant cells, while the initial, combined CT and MRI imaging techniques did not show neither osteolytic lesions, nor abnormalities of cartilage structures, as expected during an invasive neoplastic process.

On the ground of a retrospective assessment of the entire clinical follow-up and diagnostic-management procedures, a bacterial (actinomycotic) superinfection of an underlying rhinopharyngeal carcinoma becomes a reasonable conclusion, although to the best of our knowledge no literature evidences are retrievable in the international literature regarding associated rhinopharyngeal actinomycosis plus squamous cell adenocarcinoma, among HIV-infected patients too, although both disorders share some risk factors (i.e. exposure to toxic-oncogenic and infectious inhalants like tobacco, cocaine, heroin, and environmental bacteria and fungi). In our subject, it remains extremely difficult to establish whether actinomycosis overcome or not the underlying neoplastic manifestation, but while initial endoscopy-targeted biopsies addressed towards an isolated actinomycosis, only the subsequent, extensive surgical intervention and removal of the entire mass, allowed to disclose the concurrent cancer disease. Despite an apparently correct administration of an adequate antimicrobial chemotherapy, and the combined radiotherapy-chemotherapy, multiple brain secondary localizations led our patient to a rapid death.

In conclusion, our representative case report underlines that all health care professionals who are involved in the follow-up of HIV-infected patients should not overlook the possibility of multiple, overlapping, and not necessarily HIV-associated disease complications (like the extremely infrequent combination of rhinopharyngeal actinomycosis plus squamous-cell adenocarcinoma of the same rhinopharyngeal site), in order to allow a rapid recognition and a prompt and appropriate surgical and medical approach. The resort to extremely stringent techniques for collecting and submitting tissue specimens for anaerobic culture and histopathological examinations are mandatory for disclosing actinomycosis, while local invasive procedures (biopsy and histopathology studies), are needed to search an eventual, concurrent local malignancy, although an eventual superinfection may contribute to mimic and delay the recognition of an underlying cancer complication, like in our case.

**Key words:** HIV infection, rhinopharyngeal actinomycosis, rhinopharyngeal adenocarcinoma.

---

**SUMMARY**

An extremely infrequent episode of nasopharyngeal actinomycosis associated with squamous adenocarcinoma occurred in an HIV-infected male patient with a previous diagnosis of AIDS, treated with combined antiretroviral therapy taken with insufficient adherence, such that a satisfactory immune system recovery (as expressed by a CD4+ lymphocyte count persistently above 400 cells/μL), contrasted with a low-level persistence of detectable HIV viraemia, and enlarged genotypic resistance mutations. Interestingly, a number of local and specific risk factors for both infectious and neoplastic disorders were recognized by healthcare staff (tobacco smoke, long-term inhalatory substance abuse, in particular cocaine, and semi-professional mushroom-truffle hunting, including evaluation by systematic smelling). Despite appropriate and timely diagnostic assessment carried out with repeated, combined computerized tomography, magnetic resonance imaging, and fiberoptic rhinoscopy with biopsy and histopathologic studies, the final diagnosis of a combined dual infectious-neoplastic pathology occurred only after a demolishing surgical intervention and subsequent pathology studies. Despite proper antimicrobial therapy, and an associated radiotherapy and cytotoxic chemotherapy schedule, rapid dissemination of multiple secondary lesions to the brain rapidly led to our patient’s death. The imaging and histopathological diagnostics of the dual illnesses of our HIV-infected patient, and its therapeutic and outcome features, are presented and discussed on the basis of the evidence from the available literature. To the best of our knowledge, this is the first described case of actinomycosis associated with a local, underlying squamous cell adenocarcinoma of the same ear, nose, and throat district in either HIV-infected or HIV-non-infected subjects.
**RIASSUNTO**

Un episodio estremamente infrequente di associazione patologica tra actinomicosi ed adenocarcinoma squamoscellare del nasofaringe viene riportato in un paziente di sesso maschile con progressa diagnosi di AIDS, trattato con terapie antiretrovirali di combinazione assunte con aderenza insuficiente, cosicché un soddificante recupero immunologico (testimoniato da una conta di linfociti CD4+ stabilmente superiore a 400 cellule/µL), contrastato con la persistenza di viremia di HIV dimostrabile e con conseguenti, multiple mutazioni genotipiche di farmaco-resistenza. Di interesse era il puntuale rilievo da parte dell’equipe assistenziale di numerosi e specifici fattori di rischio locali per ambedue le patologie, infettiva e neoplastica (fumo di tabacco, uso prolungato di sostanze per via inalatoria, in particolare di cocaina, ed un’attività semi-professionale di ricerca di funghi e tartufi, valutati sistematicamente anche con l’odorato). Sebbene fosse stata organizzata ed eseguita una completa e puntuale sequenza di appropriate valutazioni diagnostiche, con l’ausilio di ripetute, ed associate indagini di tomografia computerizzata, di risonanza magnetica nucleare, e di rinoscopia a fibre ottiche con relativi studi bioptici ed istopatologici, la diagnosi definitiva che evidenziava una duplice patologia di origine infettiva e neoplastica si compiva soltanto dopo l’effettuazione di un intervento chirurgico demolitivo, ed i successivi studi patologici. Nonostante la somministrazione di una corretta terapia chemioantibiotica, ed un associato trattamento combinato di terapia radiante e di chemioterapia citostatica, una rapida disseminazione di multiple lesioni secondarie a livello cerebrale portava rapidamente a morte il nostro paziente. La diagnosi per immagini e lo studio istopatologico della duplice patologia del nostro paziente con concomitante infezione da HIV, insieme alle scelte terapeutiche e all’evoluzione clinica, sono alla base della nostra presentazione, e della discussione operata alla luce delle evidenze di letteratura disponibili. Per quanto ci è fino ad oggi noto, non risultano finora descritti casi di associazione tra una actinomicosi ed un locale, contemporaneo adenocarcinoma a cellule squamose dello stesso distretto orinнологico, sia in pazienti con infezione da HIV, sia in soggetti senza tale patologia concomitante.

---

**REFERENCES**


[16] Chiang C.W., Chang Y.L., Lou P.J. Actinomyco-
[17] Daamen N., Johnson J.T. Nasopharyngeal actin-
omycosis: a rare cause of nasal airway obstruction.
[18] Pradhan S., Datta N.R., Prasad K.N., Ayyagari
S., Pandey R. Actinomycosis mimicking carcinoma
[19] Özcan C., Talas D., Görür K., Aydün Ö., Yıldız A.
Actinomycosis of the middle turbinate: an unusual
cause of nasal obstruction. Eur. Arch. Otorhinolar-
[20] Chaudry S.I., Greenspan J.S. Actinomycosis in
HIV infection: a review of a rare complication. Int. J.
as a nasopharyngeal tumour: a case report. J. Laryn-
[22] Hall V., Collins M.D., Lawson P.A., Falsen E.,
Duerden B.I. Actinomyces nasicola sp. nov., isolated
from a human nose. Int. J. System. Evolution. Microbi-
[23] Che Y., Tanioka M., Matsumura Y., Kore-Eda S.,
Miyachi Y. Primary cutaneous actinomycosis of the
A., Chiodo F. Progressive intractable actinomycosis
in patients with AIDS. Scand. J. Infect. Dis. 27, 405-
Chiodo F. Invasive mycotic and actinomycotic oropharyngeal and craniofacial infection in two pa-
[26] Lee S.A., Palmer G.W., Cooney E.L. Esophageal
actinomycosis in a patient with AIDS. Yale J. Biol.
[27] García-García J.A., Corzo J.E., Ramírez M., Pine-
da J.A. Pulmonary actinomycosis secondary to bac-
[28] Tangtrakul S., Linasmita V., Wilailak S., Srisu-
pandit S., Bullngpoti S., Ayudhya N.I. An HIV-infec-
ted woman with choriocarcinoma presenting with a
[29] Sofianou D., Avgoustinakis E., Dilipoulou A, et
al. Soft-tissue abscess involving Actinomyces odontol-
yticus and two Prevotella species in an intravenous