characteristic, so diagnosis can be difficult: a lymph node biopsy is required for correct identification, since fine needle aspirates tend to be inconclusive.  

Pathogenesis remains unclear to date, but some authors have suggested that both interferon and interleukin-6 (IL-6) or cell apoptosis may play some role, pointing toward viral or autoimmune etiologies. It seems clear that KFD is an exaggerated T cell-mediated reaction to a variety of mostly infectious stimuli. The course is usually benign, and it resolves in a few months without specific treatment, although some more severe cases have occasionally been reported.

Histological findings correspond to 3 disease stages (proliferative, necrotizing and xanthomatous), representing progressive pathologic changes. The typical immunophenotype of this disease consists a predominance of CD8+ cells over CD4+ T cells. Consistent immunohistochemistry results show CD68 and CD3+, CD20+/- and CD30−. Histiococytes expressing myeloperoxidase and CD68 are characteristic of this disease.

Chest radiograph must be obtained from all patients to rule out the possibility of other causes such as neoplasms or TB. Multislice spiral tomography is of particular use in locating the most accessible lymph node for biopsy and for determining the extent of the disease.

In Peru, a country with a very high prevalence of TB, lymph node involvement is found in a good number of cases. Lymph node biopsy studies are included in diagnostic protocols, and epidemiological factors and other laboratory findings are taken into consideration. However, antituberculosis treatment is sometimes prescribed without hard scientific evidence and before the other etiological options, which would include KFD, have been explored. We therefore hold that is important to include this disease in the differential diagnosis of lymphadenopathies, particularly for lymph node TB, and appropriate studies must be conducted to avoid the prescription of costly drugs that might be unnecessary and carry significant risks for the patient.

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Actinomycosis Associated with Foreign Body Simulating Lung Cancer

To the Editor,

Pulmonary actinomycosis is a necrotizing lung infection that can develop after aspiration of a foreign body. Approximately 50% of cases can mimic lung cancer.

We report the case of a 76-year-old woman diagnosed with right lower lobe (RLL) pneumonia in September 2014, treated with azithromycin 500 mg/24 hours for 1 week. After completing the course of antibiotics, she consulted due to dyspnea accompanied by cough with foul-smelling whitish sputum and fever.

A chest computed tomography was performed (Fig. 1A), which showed heterogeneous pulmonary consolidation in the RLL, with no evidence of any obstructive central lesion. The patient received another cycle of antibiotics with amoxicillin/clavulanate acid (1000/200 tid) for 14 days.

In September 2015, she presented with hemoptysis, and fiberoptic bronchoscopy was performed, revealing a “fibrin plug in a medial subsegment of the RLL”. Bronchial angiogram revealed a hypervascularized lesion in the right hilum, irrigated by a right bronchial artery originating in an intercostal trunk. The study was completed with a PET/CT, which showed the lesion in the RLL with a maximum standardized uptake value (SUV) of 4. Given the high suspicion of malignancy and the episode of hemoptysis, we decided to perform a surgical intervention. In view of the location of the lesion, right lower lobectomy was performed by thoracotomy.

The pathology report described mixed acute and chronic inflammation, forming focal abscesses, associated with a foreign body (fish bone), with Actinomyces superinfection, fibrosis and peri-lesional reactive changes (Fig. 1B).

References

Actinomycosis is a chronic supplicative infection caused by a group of anaerobic bacteria that are normally found in the flora of oropharynx and gastrointestinal tract. Approximately 15%–20% of cases diagnosed are located in the chest. The main symptoms associated with actinomycosis are: cough (63%), hemoptysis (36%) and recurrent pneumonias (27%).\(^1\) Most patients are men, over 55 years of age, with risk factors for aspiration pneumonia, such as diabetes mellitus, alcoholism, and poor dental hygiene.\(^2\)\(^3\)

Pulmonary actinomycosis can mimic a malignant pulmonary process, so in some cases surgery is performed. Bates and Crikshank\(^4\)\(^5\) published 85 cases of pulmonary actinomycetes, of which 7 underwent lung resection due to a clinical suspicion of lung cancer.

Treatment of pulmonary actinomycosis consists of prolonged intravenous antibiotic therapy with high-dose penicillin, for 3–4 weeks. Prognosis is generally more favorable when it is diagnosed and treated early.\(^5\)

In patients with pulmonary lesions without a confirmed diagnosis of cancer, actinomycosis, even though it is rare, should feature in the differential diagnosis, particularly if there is a possibility that the patient may have aspirated a foreign body. Our case was a patient with a lung lesion caused by pulmonary actinomycosis after bronchoaspiration of a fish bone (not documented in her medical records) that mimicked a malignant process of the lung.

### References

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**Intracystic Hemorrhage in a Patient with Pulmonary Cystic Disorder Related to Light-Chain Deposition Disease**\(^7\)

**Hemorragia intraquística en paciente con afectación pulmonar quística secundaria a enfermedad por depósito de cadenas ligeras**

To the Editor,

Light-chain deposition disease (LCDD) is a rare systemic disorder characterized by the accumulation of immunoglobulin light chains in multiple organs, and is associated, in most cases, with multiple myeloma or lymphoproliferative disorders. Unlike amyloidosis, the non-fibrillar deposits are negative on Congo Red staining.\(^1\) The organs most often affected are the kidney, the heart, the liver, and the nervous system. LCDD lung involvement is very uncommon. Presentation is generally in the form of converging pulmonary cysts, although some atypical forms occur, such as nodules or bronchiectasis.\(^2\)

We report a case of pulmonary intracystic hemorrhage caused by anticoagulation in a 59-year-old woman, non-smoker, with severe pulmonary cystic involvement related with LCDD. The patient consulted due to progressive dyspnea and chest pain. In addition to her LCDD-related pulmonary cystic disease (she is on the waiting list for a lung transplant), her personal history included a diagnosis 8 years previously of multiple myeloma, and a nephrotic syndrome resistant to steroid treatment related with her LCDD. Chest radiograph (Fig. 1A) showed multiple cystic images in both lungs, predominantly in the subpleural regions (already known). Computed tomography (CT) angiogram of the chest detected pulmonary thromboembolism (PTE), and large subpleural thin-walled cystic lesions in all lobes (described in previous studies), traversed...

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