Case Report

Paucisymptomatic pulmonary and right ear tuberculosis in young woman suffering from anorexia and bulimia nervosa

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A B S T R A C T
Nowadays tuberculosis has become a reemerging infectious disease due to the many forms of immunodeficiency. Patients with eating disorders like anorexia nervosa and bulimia are a susceptible group due to the immune impairment correlated with severe malnutrition and their prevalence and incidence is growing.

We describe the case of a 31-year-old woman, with long-standing history of anorexia nervosa and bulimia, diagnosed with advanced pulmonary tuberculosis. This case underlines the importance on never neglecting even the slightest symptoms in patients with malnutrition and never excluding this pathology without a proper investigation.

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Case report

A 31-year-old middle-class Italian woman with a long-standing history of anorexia and bulimia nervosa was admitted to our hospital, in the Department of Gastroenterology, due to compromised general conditions and severe protein-energy malnutrition. In the last 6 months she lost more than 15 kg, with a body mass index (BMI) 12. She was afibrile, fatigued, alert, and orientated.

During her stay she had episodes of cough and lung CT was done.

The CT demonstrated multiple cavitations the major ones being in the superior lobes (Fig. 1) with dimensions up to 11 cm, multiple small nodules with tree-in-bud morphology and bigger nodules sparsely located in the pulmonary parenchyma suggestive for miliary locations (Figs. 1-3).

Tuberculosis was immediately suspected, and the patient was transferred to the infective disease unit and placed under respiratory isolation (Fig. 4).

Blood exams documented neutrophil leukocytosis, increase of the eritrosedimentation speed (VES) and polymerase chain reaction, and iron deficient anemia. Interferon gamma release assay was negative.

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Fig. 1 – CT axial images showing cavitations in the left and right superior pulmonary lobes and multiple miliary locations.

Fig. 2 – CT axial images showing multiple miliary locations in the medium, lingula and inferior lobes.

Fig. 3 – CT coronal image in (a) and sagittal in (b) and (c) that shows cavitation and distribution of miliary locations in the pulmonary parenchyma.
The patient developed toxic acute hepatitis due to pyrazinamide and the treatment was modified to isoniazid, rifampicin, ethambutol, and levofloxacin that was well tolerated.

Ophthalmologic evaluation there were no alteration.

A swab was taken from right ear following the insurgence of purulent secretions, pain and sensation of pressure. Microscopy showed the presence of tuberculosis.

Otolaryngologic evaluation was immediately performed and lavage therapy with acetic acid 2% was started.

Five weeks from the beginning of the therapy, with the continuous positivity of the sputum exam but with a decreasing bacterial load and an improvement of the respiratory status patient was home isolated.

Patient is currently compliant and taking the therapy.

Discussion

Tuberculosis in an important infectious disease that has been reemerging in industrialized countries for many reasons including emigration from other countries and the immunodeficiency caused by many factors. Two billion people are infected by tuberculosis be it latent or active [1,2].

Usually in the infected person the mycobacterium is contained by the immune system in a caseous granuloma but still 10%-15% of the people will develop an active disease during their life [3].

There are many risk factors that determine the infection and its latent or active state. There are factors not dependent of the individual like the droplets bacterial load, the proximity to a person with active infection, and work place especially health care workers. Others are closely related to the person and his habits. The immunity status is very important and represents a major risk factor in patients with acquired immunodeficiency syndrome (HIV), patients with immunomedi-ated diseases and in treatment with tumor necrosis factor alfa inhibitors, diabetes. Malnutrition is also an important risk factor due to the impairment of the immune systems response. Also, low socioeconomic level is seen to increase the risk of tuberculosis together with habits like smoking, alcohol consumption, and indoor pollution [4,5].

Anorexia nervosa and bulimia are important eating disorders with anorexia affecting approximately 0.3%-1% of the population [6]. The incidence of this disorder is 8/100000 persons per year and it's an upward trend [7]. There is a mean prevalence of anorexia nervosa in female especially women in their teens and young adulthood than men, with the 15-19-year-old female being more at risk and making up 40% of the cases. The incidence is much higher in this group reaching 109.2/100000 compared to the males being 1/100000 [8]. Life prevalence in females is higher and ranges from 0.9% in European countries to 2.2% in Swedish studies, and of males is 0.3% [8,9]. The prevalence of bulimia varied from 0.9% to 1.5% in women and from 0.1 to 0.5 in men [8].

These conditions are the cause of malnutrition that in many cases is severe and increases the risk of tuberculosis, even being a risk factor for the reemerging of the disease, especially in patients with BMI < 18.5, in our case the patient

Fig. 4 – CT axial images on soft tissue window showing nearly no subcutaneous fat tissue and decreased weight.

Sputum examination resulted positive for mycobacterium tuberculosis, Mantoux test was negative, blood tests for tuberculosis were negative.

Serum analysis resulted negative for HIV, HAV, HCV, and HBV vaccine-related antibodies resulted in the range.

A four-drug therapy was immediately started using isoniazid, rifampin, ethambutol, pyrazinamide.
had a BMI of 12 in the moment of admission [10,11]. Severe malnutrition, especially protein-energy malnutrition, causes immunity impairment especially cell-mediated immunity especially the delayed type hypersensitivity, that is the main defense mechanism of the host toward tuberculosis. It alters T-cell population and the CD 4/CD 8 ratio, lowers their peripheral numbers, and even impairs their ability to react [6,12].

França TGD et al. even referred a much higher yearly prevalence of tuberculosis 260.2/10,000 in the underweight compared to 24.7/100,000 in the normal weight cases [13].

The first step in diagnosing tuberculosis is the clinical suspect based on signs and symptoms.

The most frequent ones are: cough for more than two weeks, productive cough, fatigue, fever, weight loss, night sweat, chest pain, hemoptysis. After the first suspect usually, the first diagnostic exam is chest x-ray which is not very specific.

Microscopy has been the cornerstone of the diagnosis. Sputum smear microscopy for acid fast bacilli is able to identify most cases of tuberculosis, 3 Acid-Fast Bacilli (AFB) smears have a sensitivity for pulmonary TB of approximately 70%. Collecting sputum early in the morning increases possibility of the diagnosis. Mycobacterial culture is the golden diagnosis for tuberculosis diagnosis.

Newer techniques have improved the possibility of diagnosis. Interferon gamma release assays (IGRAs) were a more specific test that would not contain antigens from bacilli.

Fluorescence has improved much with the new microscopes having achieved 10% more sensitivity than Ziehl-Neelsen. Newer liquid media systems based on fluorometric growth indicators that have increased yield of 10% are quicker to identify tuberculosis than the classic solid ones.

Polymerase chain reaction (PCR) is an important development in the diagnosis of tuberculosis being a fast technique with high specificity and sensitivity. It can be also used with the new line probe assay that augments its potential increasing its usefulness [14,15].

Imaging techniques like CT play an important role in the diagnosis of tuberculosis and in determining cavitations, miliary form, or complications. CT can correctly diagnose tuberculosis in 91% of the patients and exclude it in 76% which is far more superior than the chest x-ray [16].

First line treatment of tuberculosis consists of isoniazid, rifampicin, pyrazinamide, ethambutol, streptomycin but can be modified due to hepatoxicity and drugs like levofloxacin can be utilized.

Patients treatment should last 6 months. During the first 2 months isoniazid, rifampicin, pyrazinamide, and ethambutol should be used and the following 4 months isoniazid and rifampicin should be used [17].

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