Myelofibrosis with Tuberculosis: A Case Report

YK Jin, QL Shi, CH Peng, H Yu, XY Zhang, XW Ye

Guizhou Provincial People’s hospital, Guiyang, China

Abstract  Myelofibrosis (MF), or fibrosis of the bone marrow, is an uncommon condition. Most cases arise secondary to other disease processes. However, whether tuberculosis can stimulate a secondary fibrotic reaction or develop in patients who have preexisting myeloproliferative disorders is not clear. Here we present a case of a 54-year-old man whose myelofibrosis disappeared completely after administration of antituberculous treatment. The purpose of reporting this case is to reconsider the relationship between tuberculosis and secondary myelofibrosis.

Keywords: myelofibrosis, tuberculosis


1. Case Report

A 54 year old man came to our hospital with complaints of repeated fever, chronic cough, sweating and general pain for over 20 days. He had a history of pulmonary tuberculosis (TB) a year ago, because of the discomfort of gastrointestinal, he stopped the drugs of antituberculosis himself after using only one month. He had no underlying debilitating disorders. On physical examination, the patient had a fever of 38.5°C, with a left supraclavicular sternum tenderness. Examination of the respiratory system showed normal breath sounds without moist rales or rhonchi on both lung fields. Other systems were normal.

The chest CT showed that the distribution of the diffuse miliary nodules in both lung, enlargement of hilar lymphatic nodules. Thoracic uneven bone density, bone destruction in part. The examination of marrow cell showed anemia and nucleated cell proliferation activity, the neutrophils ratio increased obviously, about 73%. Bone marrow biopsy presented myelofibrosis. Ultrasound of heart and abdomen was negative. Both colonoscopy and gastroscope didn’t show abnormal. Head CT plain scan was negative. Several active bone metabolism in the whole body were found by skeleton ECT scan, its property was undefined. Routine blood showed severe anemia repeatedly (HGB:50-60g/L). Based on the history of tuberculosis and the examinations the patient had, anti-TB treatment was started with 2HRZE/ 6HR. With this treatment he showed a phenomenal improvement after 10 days. His general pain relieved, and fever also subsided. About 4 months later, his bone marrow biopsy did not showed myelofibrosis any more.

2. Discussion

Myelofibrosis (MF), alternatively is also known as myelosclerosis, is a rare hematologic disorder characterized by extensive fibrosis of the bone marrow. In a majority of cases, the etiology of this disease is unknown. However, many conditions such as extensive granulomatous or neoplastic involvement of the bone marrow, chronic irradiation, as well as benzene or phosphorus poisoning have been suggested as possible causes. [1] When it appears as a histological feature associated with well-characterized malignant or benign diseases of hematopoietic or non-hematopoietic origin, is considered to be a secondary disorder. Among non-hematopoietic diseases that occur concomitantly with myelofibrosis, tuberculosis is reported sufficiently frequently to raise the possibility that a relationship exists between the 2 entities. [2,3] An association between myeloproliferative disorders and tuberculosis was first postulated to exist over 70 years ago. [4] Several reports have focused on tubercular infections in patients with preexisting chronic myeloproliferative disorders associated with myelofibrosis, such as myeloid metaplasia. [5] Large autopsy studies have indicated that the frequency of tuberculosis is 2-2.5-fold higher worldwide among patients with myelofibrosis or chronic myelogenous leukemia than in the general population. [6] Recent study in vitro models showed that exposure to a mycobacterial antigen, ESAT-6, exacerbates granulomatous and fibrotic changes. [7] Verma et al found that Mycobacterium tuberculosis induced IL-1 secretion and pro-fibrotic responses by fibroblasts. [8] In a case report, present a 73-year-old man with preexisting myeloproliferative disorder, combined with tuberculosis. This combination of complications exacerbates the disease, leading rapid leukemic transformation and the patient’s death. [9]

Allogeneic stem cell transplantation is the only treatment with a curative potential, but only a minority of patients are eligible for it. [10,11] Bone marrow transplantation, cord blood transplantation are curative in some cases probably. [12,13] Many asymptomatic patients, if stable, do not require treatment. Therapy is directed to manage complications and to improve quality of life. For
younger patients allogeneic marrow transplantation should be considered. [11] Other therapeutic options are Corticosteroids, IV Immunoglobulins, Alfa Interferon, which may lead to resolution of fibrosis and restoration of blood counts. [14] Use of Hydroxyurea, Thalidomide, alone, or in combination with Prednisolone are other options. [15] The identification of cause like Tuberculosis should prompt treatment of that disorder. Whether an interaction between tuberculosis and myelofibrosis exists was moot, the clinical history of our patient suggests that a causal relationship exists between the 2 entities, because myelofibrosis was not observed in the bone marrow biopsy section after antituberculous treatment was administered. This strongly suggests that a causal relationship did exists between tuberculosis and myelofibrosis. Physician should be aware of this association, and testing for tuberculosis should always done for patients with myelofibrosis.

References


