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Case Report
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Abdominal Tuberculosis Masquerading as Progression of Carcinoma Prostate

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Abstract

The presence of pulmonary tuberculosis in cancer patients is a common entity occurring due to the immunosuppression by the cancer itself or secondary immunosuppression from chemotherapy. While there have been many reported cases of pulmonary tuberculosis, the occurrence of abdominal tuberculosis in urologic cancers has not been documented in literature. Here we present the case of a 64 year old male, a known case of castration resistant carcinoma prostate with skeletal metastasis, 10 weeks post chemotherapy, who presented with complaints of abdominal distension for 3 weeks with loss of weight, pedal edema, reduced appetite and generalized body weakness, which was assumed to be disease progression but later diagnosed as abdominal tuberculosis. We hope to highlight the possibility of abdominal tuberculosis in the scenario of urological cancers.

Introduction

Tuberculosis (TB) remains the most ubiquitous infectious disease worldwide and leads to high mortality. The World Health Organization (WHO) estimates that 1.8 billion people- close to one quarter of the world's population- are infected with Mycobacterium tuberculosis. Globally, an estimated 10 million people fell ill with TB in 2019, a number that has been slowly declining in recent years. There were an estimated 1.2 million TB deaths among HIV negative people and an additional 208,000 deaths among HIV positive people [1].

Chemotherapy administration is a risk factor for occurrence of infection, of which one is tuberculosis. There are a number of mechanisms that lead to the occurrence of tuberculosis in patients with cancer, which includes impairment of the immune response due to malignancy per se. or secondary immunosuppression due to effects of cytotoxic chemotherapy.

A multiple number of cases of such infections have been reported. Most commonly pulmonary tuberculosis occurs in patients on or immediately following chemotherapy. TB may occur as a primary foci or reactivation of the dormant foci. While there have been many case reports of such cases, there has not been any reports of abdominal TB post cancer chemotherapy.

Case Presentation

A 64 year old male, known case of castration resistant carcinoma prostate with skeletal metastasis post TURP and 10 weeks post-docetaxel chemotherapy, came with complaints of abdominal distension for 3 weeks with significant loss of weight, reduced appetite and generalized body weakness. He has no significant personal or family history. He has no history of drug allergy.

On examination, the patient was stable with the following vital signs: Blood pressure 110/70 mmHg, Heart rate 80/min, Respiratory rate 24/min, Temperature 98°F. He had anasarca. Examination of the respiratory and cardiovascular system was essentially normal. Local examination of the abdomen revealed that it was soft, distended with the presence of free fluid. Laboratory investigation done revealed creatinine 1.1 mg/dl, Potassium 4.8 mEq/L, Sodium 125 mEq/L, Total WBC 5300 cells/cumm, basophils 0%, eosinophils 0%, lymphocytes 25%, monocytes 3%, polymorphs 72%, Hb 9 gm/dl, platelet count 261,000 cells/μL. Urine analysis was normal and urine and blood culture were negative.

The patient was admitted under the oncology team with suspicion of disease progression and was undergoing supportive care. The patient developed fever on the 3rd day following admission and was advised for CT chest and abdomen. The CT revealed diffuse peritoneal and omental inflammation with moderate ascites and mediastinal necrotic adenopathy, which is suggestive of infective etiology, and less likely metastatic disease. A probable diagnosis of abdominal tuberculosis was made and

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the patient underwent diagnostic laparoscopy with peritoneal and omental biopsy. Histopathology of the peritoneal biopsy revealed necrotizing granulomatous inflammation, suggestive of tuberculosis. The patient was started on ATT following the biopsy results. On the consequent follow ups the patient's symptoms gradually improved over time.

Discussion

The reactivation of tuberculosis is a known adverse effect of chemotherapy. There have been case reports of reactivation of pulmonary tuberculosis following Docetaxel chemotherapy; however peritoneal tuberculosis following Docetaxel chemotherapy has not been documented.

Approximately 5% to 10% of the individuals infected with *Mycobacterium tuberculosis* (*M. tuberculosis*) develop the disease during the first 2 to 5 years after infection [2]. In the rest of them, the innate immune response will either fully eliminate the infection without leaving a trace of immunological response (resistance to TB infection) [3] or lead to a state of persistent immune response to *M. tuberculosis* antigens without clinical evidence of active disease [3,4]. This last outcome is indeed the basis to consider that one fourth of the world population is infected with *M. tuberculosis* [5]. These are the individuals who persist with the so-called Latent Tuberculosis Infection (LTBI) immunoreactivity even if bacterial clearance is achieved. The dominant paradigm for LTBI has been one of a fine balance between host immune response and pathogen metabolism so that progression to active disease occurs whenever this balance is disrupted. In persons with LTBI, several factors increase the risk of developing active TB, one of which is cancer.

The incidence of TB has been increasing in patients with cancer, in both pulmonary and non-pulmonary cancer [6-8]. Also the risk of TB reactivation reasonably increases in people with cancer. Moreover, a diagnosis of TB should be ruled out in this high risk group, and therefore screening for active and latent TB should be done [9], which may either pulmonary or extra-pulmonary. Although considering the ubiquitous exposure to tuberculosis for the Indian population, the applicability of tuberculin testing may be inconsequential, the newer generation Interferon Gamma Release Assay (IGRA) may prove to be helpful.

The IGRA is a whole blood assay to detect the IFN-γ produced in vivo by sensitized T cells after *in vitro* stimulation with mycobacterial antigens. The mycobacterial antigens used in these assays are the Early Secretory Antigenic Target (ESAT-6) and the 10-kDa Culture Filtrate Protein (CFP-10) ESAT-6 and CFP-10 antigens are encoded in the Region of Differentiation 1 (RD1) present in the *M. tuberculosis* and *Mycobacterium bovis* genome and are absent in the Bacillus Calmette-Guerin vaccine (BCG) and most environmental mycobacteria [10,11]. Therefore, IGRA results are affected by neither BCG vaccination nor exposure to environmental mycobacteria. However, their precision is low in immune-compromised individuals being screened for LTBI, which may hamper its widespread applicability.

Our patient had no history of tuberculosis, and did not show any signs of opportunistic infections post chemotherapy. The initial presentation of Anasarca with abdominal distention and failure to thrive was attributed to disease progression and the patient was advised to undergo further chemotherapy once general condition improved with supportive care. But the development of fever prompted us to investigate, with CT showing suspicious infective etiology, later clinching the diagnosis. Our patient was not evaluated for latent tuberculosis infection, which may have helped us counsel them to report earlier if any signs of ill health were present.

The occurrence of tuberculosis in urological cancers is low (336 per 1 lakh population), and hence the diagnosis is usually delayed unless strong suspicion arises [12]. Highlighting our experience with this case we would like to recommend consideration of screening IGRA tests in patients who are to be started on chemotherapy.

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