JMSCR Vol||11||Issue||11||Page 96-99||November 2023

http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v11i11.14

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

A Rare Coexistence of Cervical Node Tuberculosis in CA Pancreas

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Abstract

The association of TB with carcinoma was initially described ~200 years ago by Bayle who considered 'cavitation cancereuse' as one of the various types of TB(1) Since both disorders are common, we support the notion that in most cases their co-existence may be explained by chance alone.

Nowadays, research for carcinogenesis is expanding and the possible correlation between chronic inflammation and cancer development is slowly being unraveled.

Although TB and cancer are very common diseases, there has been little attention to the pathophysiological and practical implications of their co-existence. We sought to review the available evidence and identify the data that refer to the correlation between TB and malignancy in order to highlight neglected aspects of this association and probably derive clinically useful information.

Introduction

Tuberculosis (TB) presents a global threat in developing and developed countries. According to the World Health Organization, over 2 billion people, equal to one-third of the world's population, are infected with tuberculosis bacilli. On the other hand, cancer is the second leading cause of death after coronary artery disease with 11.3 million new cases and 7.9 million deaths (~13% of all deaths)(2). Population with malignancy is growing worldwide; however, its tuberculosis (TB) burden including remains unclear regarding incidence, mortality, and relapse (3).

In addition, due to advancements in current medications, the average lifespan after a diagnosis of cancer is longer than before. The risk for TB in patients with malignancy is due to immunosuppression from the cancer itself or from

the chemotherapy and local structural changes in the lungs by primary lung cancer or metastasis.

Simultaneous carcinoma and tuberculosis cases are rarely encountered in other sites such as the colon, stomach, peripancreatic node, breast, and buccal mucosa.

In this case, we will be discussing a patient with pancreatic cancer who has developed tuberculosis of the cervical lymph nodes which is a rare occurrence.

Case Report

A 59-year-old female patient k/c/o periampullary carcinoma,

Status post: Whipple's procedure.

- Presented with complaints of swelling in the bilateral supraclavicular region for 6 months, associated with fever, cough without expectoration, history of weight loss, occasional night sweats and fatigue, aggravated since the last 20 days.
- Swelling was insidious in nature, gradually increased in size, and localised tenderness was present.
- Fever was gradual onset, aggravated in the nights showing diurnal variation.
- Cough was gradual onset, not associated with expectoration aggravated on exertion and relieved on rest.
- Not associated with expectoration, chest pain, hemoptysis, dyspnea.
- General Examination: Patient was conscious, coherent, and cooperative, patient appears to be moderately built, Temp: 101°f, PR: 99 bpm, RR: 22 cpm, SPO2: 99% in RA, BP: 130/80 mmHg.
- O/E : CVS : S1+ S2+, RS: B/L NVBS+, with no added sounds.
- CHEST X-RAY: Showed widening of the mediastinum, no obliteration of the costo-phrenic angles and no other abnormality was noted in the lung parenchyma.
- Blood investigation: CBC showed decreased hb: 9gm/dl, LFT was deranged, ESR & CRP were elevated,
- PET-CT scan findings show - a small volume of mildly avid and non-avid bilateral level IV and supraclavicular lymph nodes. Mild metabolic and morphologic progression of mediastinal lymph nodes. Hepatomegaly with mild ascites.

The supraclavicular largest right lymph node now measures 11x8mm with an SUV max of 3.19 (previous 10x7mm with SUV max of 1.72)

- The patient underwent a biopsy of the supraclavicular lymph node and was started on symptomatic treatment.
- Biopsy report - Gross examination: Nodular fibrofatty tissue measuring 3.0 x 1.5 x 1.0 cm. Microscopic examination shows fragments with coalescent granulomas composed of epitheloid cell clusters surrounded by dense lymphocytic infiltrate, areas of necrosis and Langhan's giant cells noted. Impression: Consistent inflammation may be of Koch's etiology.

Discussion

The treatment protocol for patients with lymph node tuberculosis is - 2 months of intensive phase R (Rifampicin), H (Isoniazid), Z (Pyrazinamide), E (Ethambutol) f/b 4 months of continuous phase - R (Rifampicin), H (Isoniazid), E (Ethambutol), six months of ATT is the standard first-line regimen is recommended for peripheral lymph node TB [(4)].

In patients whose condition has worsened or deteriorated along with immunosuppression, it is recommended to extend the regimen for an additional 3 months of RHE f/b biopsy sent for histology and TB culture in patients who fail to respond [(5)].

Regular follow-up of the patient should be done during the course of treatment with repeat lung function tests, monthly in order to prevent further damage and intervene the medical management if necessary.

The risk of TB reactivation reasonably increases in people with cancer, and therefore, screening for

active and latent TB in this group should be considered. To prevent relapse, the patient can be treated for tuberculosis for a duration of a year.

Besides the well-known increased TB risk in patients with malignant disorders that is secondary to the immunosuppressive effects of some of the implemented therapies (i.e. steroids) or the associated immune dysfunction (i.e. in lymphomas), the main findings of this review are the following: (i) TB infection may be associated with the subsequent development of cancer; (ii) TB and malignancy may co-exist in some cases; and (iii) similarities in the clinical and radiological presentations between TB and malignancy might mislead diagnosis[6].

Of note, malignancies and the instituted therapies for their management create the proper environment for either the reactivation of a latent TB infection or, more rarely, for the acquisition of a primary mycobacterial infection. Immunosuppression, especially depression of the T-cell defence mechanisms, is associated with mycobacterial infections.

In general, chronic inflammatory conditions have been thought to create the appropriate microenvironment for malignancy development through a number of mechanisms; i.e. the higher rate of cell turnover is thought to increase the risk for genetic errors [7].

Mycobacterial infections may escape the host's cellular response, and establish chronic and persistent inflammation. There is ample experimental evidence that *Mycobacterium tuberculosis* is capable of inducing DNA damage. Specifically, various mycobacterial cell wall components may induce the production of nitric oxide, and reactive oxygen species. It should be noted at this point that nitrative DNA damage as well as oxidative DNA damage have been implicated in inflammation-related carcinogenesis [8] .

Recent data reveal that *M. tuberculosis* may also enhance synthesis of BCL-2 and this could lead to increased antiapoptotic activity. Some clinical and experimental data suggest that mycobacterial infections are associated with elevated concentrations of leukotrienes and prostaglandins. In addition, vascular endothelial growth factor, a mediator with significant angiogenic, mitogenic and vascular permeability-enhancing properties has been shown to be secreted by peripheral blood mononuclear cells incubated with purified protein derivative of tuberculin and correlates with cavity formation in active pulmonary TB[9].

The combination of direct DNA damage, apoptosis inhibition and perpetuation of chronic inflammation may enhance mutagenesis of progeny cells and these effects, coupled with enhanced angiogenesis, may lead to a microenvironment that is highly conducive to tumorigenesis [10].

Conclusion

The relationship between TB and malignancy becomes even more challenging to be clarified when two other parameters are taken into consideration. First, several studies have reported on tissue biopsies where tuberculous inflammatory changes and malignant cells co-existed. Furthermore, although new radiological imaging studies such as combined positron emission tomography (PET) and computed tomography (CT) have enabled clinicians to make a more accurate diagnosis, the ability of these disorders to clinically mimic one another may present a serious challenge in the establishment of the diagnosis[11].

Along these lines, our data strongly suggest that TB and various types of malignancies are able to mimic each other and have atypical clinical and radiological expressions. Misdiagnosis may be detrimental; in the case of an underlying or co-existent tuberculous infection, the commencement of immunosuppressive therapy may lead to dissemination and often fatal infection.

In conclusion, the other perspectives of TB-related burdens, including mortality and recurrence, should be investigated for evaluating the prognosis in patients with malignancy; however,

these perspectives are yet to be researched. Clinicians need to be aware of the myriad manifestations of TB and resist the temptation of premature diagnostic closure. The diagnosis of a tuberculous infection remains challenging and requires a high index of suspicion, especially when it complicates the clinical presentation of cancer patients. Also, further research is warranted to determine if a tuberculous infection, similar to other chronic infections and inflammatory conditions, may facilitate carcinogenesis.

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