Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2023; 2(2)

Diagnosis of Tuberculosis infection in HIV: A Review

Hendra Wahyudi[1*]
[1] *Department of Internal Medicine, Ibnu Sina General Hospital, Aceh Besar, Indonesia*

***Corresponding Author**: Hendra Wahyudi, E-mail: hendra@gmail.com

ARTICLE INFO

Article history:
Received
11 December 2022

Accepted
28 February 2023

Manuscript ID:
JSOCMED-111222-22-5

Checked for Plagiarism: Yes

Language Editor:
Rebecca

Editor-Chief:
Prof. Aznan Lelo, PhD

ABSTRACT

The high prevalence and increasing incidence of HIV has adversely affected the control of several endemic diseases, including tuberculosis (TB). In HIV there is a progressive decrease in CD4 T cells which is associated with progressive damage to immunity, in the form of a severe decrease in digestive tract lymphoid cells, erythrocyte apoptosis, increased permeability of the digestive tract and finally massive CD4 T cell destruction. HIV will attempt to enter target cells (dendrite cells, macrophage cells), which are cells capable of expressing CD4 T cell receptors and express chemokine coreceptors (CCR5 or CXCR4) on the surface of CD4 T cells. HIV utilises CXCR4 to destroy CD4 T cells at acute onset, resulting in a decrease in CD4 T cell numbers. The condition of decreased CD4 T cell count in HIV will also be aggravated by the presence of TB co-infection. CD4 T cells contribute to controlling Mycobacterium tuberculosis. The adaptation of Mycobacterium tuberculosis in HIV patients can also weaken the cytokine immunity of interfor-γ, interleukin-10 patients in HIV infection. HIV-1 induces a decrease in CD4+ levels and the development of active tuberculosis. This review summarizes that *Mycobacterium tuberculosis* has an important component, Lipoarabinomannan (LAM), which has a broad ability to inhibit the influence of immunoregulators, thereby suppressing the proliferation of T lymphocytes, inhibiting macrophage activation and neutralising the influence of free radicals. Decreased immune status and nutritional status due to *Mycobacterium tuberculosis* can accelerate the course of HIV infection towards AIDS.

Keywords

Mycobacterium tuberculosis, HIV infection, AIDS.

How to cite: Wahyudi H. Diagnosis of Tuberculosis infection in HIV: A Review. *Journal of Society Medicine*. 2023;2(2): 54-57. **DOI: https://doi.org/10.71197/jsocmed.v2i2.64**

INTRODUCTION

Mycobacterium tuberculosis or TB bacilli spread to the hilus lymph nodes. The first lesion in the alveolus, infection of the lymph nodes and associated lymphadenitis form the primary complex. Once in the lymph nodes, the TB bacillus can spread through the lymph ducts and bloodstream to other organs such as the liver, lymph nodes, kidneys, bones, brain and others [1].

TB bacilli can cause disease directly in organs or live dormant in tissue macrophages and can cause active TB years later. Tubercles can also disappear with resolution, calcify to form Ghon complexes, or necrosis with lentil material formed from macrophages. If the lentil material liquefies, the bacilli can multiply extracellularly and expand in the lung tissue and can spread gradually to cause lesions in other organs, known as miliary TB [1,2].

The immune response consists of delayed type hypersensitivity (DTH) and cell-mediated immunity (CMI) that will occur within 4 to 6 weeks after primary infection. Antigens process antigen presenting cells (APCs) to produce major histocompability complex (MHC). There are two classes of MHC: T cells that assist immune/T-helper function known as CD4 belong to MHC class II and T cells that function as suppressors or

cytotoxic known as CD8 are associated with MHC class I. The body's resistance to TB depends on CD4 function, where if there is a CD4 deficiency, the individual will be susceptible to TB infection.

DIAGNOSIS OF TUBERCULOSIS INFECTION IN HIV

1. Clinical Screening Alogarithm

WHO recommends screening for TB at the time HIV infection is diagnosed, before starting anti-retroviral therapy and periodically during follow-up. There is currently no internationally accepted screening for TB. Several studies have been conducted to develop simple methods for TB in patients with HIV infection.

2. Radiological examination

The spectrum of radiographic manifestations of pulmonary TB depends on the relative level of immunodeficiency in the HIV patient. During the early phase, when HIV patients are not immunosuppressed, the radiographic pattern is similar to non-HIV-infected individuals with more typical lesions of superior lobe infiltrates with or without cavities [3,4]. The addition of chest x-rays for symptomatic screening increases the number of TB cases detected but is non-specific and adds to the cost burden. Chest x-rays may still be proportionally incorrect in diagnosing TB in patients with HIV immunosuppression. In addition, chest x-rays may appear normal in 7-14% of HIV/TB patients [3].

3. Sputum BTA

The sputum bacilli resistant acid (BTA) method is the most common method of TB detection. Microscopy examination has the advantages of being inexpensive, relatively quick to perform, and specific. However, it must be considered that a positive specimen result should contain approximately 105 mycobacteria per millilitre. The sensitivity of sputum BTA in patients with HIV infection is about 43-51%. Methods that increase the speed or sensitivity of sputum microscopy are fluorescence microscopy and alternative specimen processing methods, such as concentrating, sedimenting and morning sputum collection. These procedures can increase the sensitivity by 13-33% on microscopic examination, but culture examination is used as the standard reference [3].

4. Culture

Culture of Mycobacterium tuberculosis is much more sensitive than microscopic staining and has been recommended to assist in the diagnosis of TB with HIV infection. Culture also allows characterisation and drug susceptibility testing. Solid media inoculsion methods such as Lowenstein-Jenson (L-J) media or Middlebrook media show sensitive but slow results, with growth around 6-8 weeks of incubation. This leads to delays in initiating therapy, with detrimental effects on TB with HIV co-infection. Automated liquid culture systems can detect tuberculosis microbacterium growth within 1-2 weeks with Bacterial carbon dioxide production or oxygen consumption with radiometric sensors (BACTEC 460 TB; Becton Dickinson Diagnostic Instruments Systems, USA), fluorescent sensors, colourimetric sensors, pressure sensors [5-7].

5. Molecular Techniques

Nucleic acid amplification testing (NAAT) is a reliable technique to improve diagnosis. Commercial kits have the advantage of good standardisation. This technique has a sensitivity of more than 95% and specificity of 100% [6-9].

6. GeneXpert-Rif

The WHO endorses the use of GeneXpert-Rif for rapid diagnosis of TB as well as rifampicin resistance in HIV patients with clinical TB suspicion. GeneXpert is a TB-specific automated, cartridge-based nucleic acid amplification assay, has integrated and automated sample preparation, real-time PCR amplification and

detection, and provides results in less than 100 minutes. Clinical validation trials were conducted in four phases, showing results of 92.2% of positive cultures detected by a single Xpert MTB/RIF test. The test thus has the potential to complement current reference standard TB diagnostics and improve overall sensitivity [7,10].

CONCLUSION

This review summarizes that Mycobacterium tuberculosis has an important component, Lipoarabinomannan (LAM), which has a broad ability to inhibit the influence of immunoregulators, thereby suppressing the proliferation of T lymphocytes, inhibiting macrophage activation and neutralising the influence of free radicals. Decreased immune status and nutritional status due to Mycobacterium tuberculosis can accelerate the course of HIV infection towards AIDS.

DECLARATIONS

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

FUNDING

This research has received no external funding.

COMPETING INTERESTS

None.

AUTHORS' CONTRIBUTIONS

All authors significantly contribute to the work reported, whether in the conception, execution, acquisition of data, analysis, and interpretation, or in all these areas. Contribute to drafting, revising, or critically reviewing the article. Approved the final version to be published, agreed on the journal to be submitted, and agreed to be accountable for all aspects of the work.

ACKNOWLEDGMENTS

None

REFERENCE

1. Depkes RI. Pedoman Nasional Penanggulangan Tuberkulosis. In Jakarta; 2011. p. 12–25.
2. Aditama T. Tuberkulosis dan AIDS in Tuberkulosis: Diagnosa, terapi dan masalahnya. In Jakarta: Yayasan Penerbit Ikatan Dokter Indonesia; 2005. p. 80–8.
3. Padmapriyadarsini C, Narendran G, Swaminathan S. Diagnosis & treatment of tuberculosis in HIV co-infected patients. Indian J Med Res. 2011; 134 (6): 850–65.
4. Kistan J, Laher F, Otwombe K, Panchia R, Mawaka N, Lebina L, et al. Pulmonary TB: varying radiological presentations in individuals with HIV in Soweto, South Africa. Trans R Soc Trop Med Hyg. 2017; 111 (3): 132–6.
5. Daftary A, Calzavara L, Padayatchi N. The contrasting cultures of HIV and tuberculosis care. AIDS. 2015; 29 (1): 1–4.
6. Niemz A, Boyle DS. Nucleic acid testing for tuberculosis at the point-of-care in high-burden countries. Expert Rev Mol Diagn. 2012; 12 (7): 687–701.

7. Denkinger CM, Schumacher SG, Boehme CC, Dendukuri N, Pai M, Steingart KR. Xpert MTB/RIF assay for the diagnosis of extrapulmonary tuberculosis: a systematic review and meta-analysis. Eur Respir J. 2014; 44 (2):1-5.
8. Luckheeram RV, Zhou R, Verma AD, Xia B, Luckheeram RV, Zhou R, et al. CD4 + T Cells: Differentiation and Functions. Clin Dev Immunol. 2012; 1: 1–12.
9. Nasronudin. Penatalaksanaan Koinfeksi Penderita HIV: HIV AIDS, Pendekatan Biologi Molekuler, Klinis dan Sosial. In: 2nd ed. Surabaya: Airlangga University Press; 2014. p. 239–47.
10. Philips JA, Ernst JD. Tuberculosis Pathogenesis and Immunity. Annu Rev Pathol Mech Dis. 2012; 7 (1): 353–84.