

Abnormalities of liver function tests in a patient with tuberculosis on antituberculous treatment

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ABSTRACT

Background: Tuberculosis (TB) remains a noteworthy worldwide medical issue even though the causative living being was found over 120 years prior and profoundly compelling medications and antibody are accessible making TB a preventable and treatable malady. India is the most astounding TB load nation on the planet. It represents almost one-fifth of the worldwide weight of TB. Consistently, all-inclusive around 18 lakh people create TB, of which around 8 lakh are new smear constructive very irresistible cases and about 4.17 lakh individuals pass on of TB; consistently, two people kick the bucket each 3 min and around 1000 individuals pass on consistently. **Methodology:** Patients liver function will be assessed by measuring serum bilirubin, serum alkaline phosphatase (SAP), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), serum proteins, and prothrombin time. **Results:** Out of 50 patients on anti-TB treatment, four patients had increased serum bilirubin, SGOT, and SGPT. All these four patients are chronic alcoholic. **Conclusion:** The management of drug-induced liver disease and active TB in these patients is difficult and the hepatotoxicity of anti-TB drugs is enhanced in transplant patients.

KEY WORDS: Liver dysfunction, Pathogenesis, Tuberculosis

INTRODUCTION

Tuberculosis (TB) is a bacterial infection caused by the acid-fast bacilli *Mycobacterium TB*. The principle lesions are usually in the lungs. Other organs such as lymph nodes, intestines, kidney, bone, and meninges are also involved through dissemination.^[1]

The portal of entry of this organism is almost exclusively through the lungs (droplet infection) 3000 infectious nuclei per cough except in bovine TB, in which the portal of entry is oral.^[2]

TB affects human beings in two forms;

1. Primary infection – in which tubercle bacilli invade the host that has no specific immunity
2. Secondary form (reinfection or adult TB) – in which the bacilli produce the disease in the phase of specific immunity.

Pathogenesis

The pathogenesis depends on the development of antimicrobial cell-mediated immunity. This is

determined by distinctive features of the bacilli and host factors. Tubercle bacillus has three important features which distinguish it from most other pathogens and also determines the course of the disease.^[3,4] These are

1. Slow generation time
2. High lipid content of the bacillus
3. Lack of either exotoxin or endotoxin.

The hallmark of TB lesion is caseating necrosis with varying degrees of exudation, Langerhans giant cells, tubercle formation, and fibrosis.

Pharmacotherapy of TB

The only disadvantage is high cost of short-term chemotherapy.

There are number of short-term treatment of 6 months duration that is highly effective with low toxicity. The regimens used in directly observed therapy short course in the Revised National TB Control Program in India.^[5,6]

Antituberculous Agents^[7-10]

First-line antituberculous agents for treatment of susceptible TB consist of isoniazid, a rifamycin, ethambutol, and pyrazinamide. Some of the second line

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Isoniazid	Common	Anorexia, nausea, vomiting, fever, skin rashes, peripheral neuropathy
	Rare	Hepatitis, Vertigo, convulsions, atrophy, hemolytic anemia, pellagra, hyperglycemia arthralgia
Rifampicin	Common	Orange-red discoloration of urine, anorexia, nausea, vomiting, diarrhea, skin rashes
	Rare	Flu-like syndromes, hepatitis dyspnea, hypotension, menstrual disturbances, muscular weakness
Pyrazinamide	Common	Anorexia, nausea, vomiting, fever, hepatitis, skin rashes, arthralgia
	Rare	Sideroblastic anemia, photosensitization, gout, dysuria
Ethambutol	Common	Optic neuritis, arthralgia
	Rare	Hepatitis, interstitial nephritis

anti-TB drugs are amikacin, kanamycin, capreomycin, ciprofloxacin, thioamide, ethionamides etc.

MATERIALS AND METHODS

Pre-treatment Evaluation

Patients are well informed and consent will be taken from the patients before their enrolment in the study and their compliance solicited. They will be advised not to stop the prescribed medications for any reason on their own. They will be asked to report immediately if any adverse symptoms are noticed such as nausea, vomiting, loss of appetite, yellowish discoloration of skin, and mucous membrane. Patients liver function will be assessed by measuring serum bilirubin, serum alkaline phosphatase (SAP), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), serum proteins, and prothrombin time. The liver function tests will be done before drug treatment and then at 1st, 2nd, 4th, 8th, 12th, 16th, 20th, and 24th weeks of treatment.^[11-13]

The following investigations will be performed before starting chemotherapy in patients of newly discovered pulmonary TB.

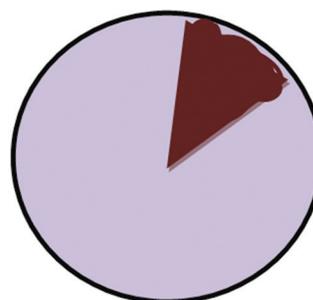
1. Hb%
2. Platelet count
3. Serum proteins – total and albumin
4. Serum bilirubin – total and direct
5. SAP
6. SGOT
7. SGPT
8. Prothrombin time
9. Chest X-ray PA view
10. GGT.

RESULTS AND DISCUSSION

Out of 50 patients on anti-TB treatment, four patients had increased serum bilirubin, SGOT, and SGPT. All these four patients are chronic alcoholic, who absence per alcohol after antituberculous therapy (ATT). Hence, there is a correlation between alcoholic and liver dysfunction patients who are on ATT.

Patients with increased liver enzymes on antituberculous therapy

Patients without increased liver enzymes on antituberculous therapy



Patients with increased Liver enzymes on ATT.

Patients without Increased Liver enzymes on ATT

CONCLUSION

The management of drug-induced liver disease and active TB in these patients is difficult, and the choice of ATT early after liver transplantation remains controversial.^[6,7] In addition, the hepatotoxicity of anti-TB drugs is enhanced in transplant patients, mainly due to the function of the graft and interactions with immunosuppressive drugs.

REFERENCES

1. Park K. Park's Textbook of Preventive and Social medicine. 24th ed. Jabalpur: Bhanot Publishers; 2017. p. 146-55.
2. Government of India. Annual Report 2008-2009. New Delhi: Ministry of Health and Family Welfare; 2004.
3. Maitra A, Kumar V. Robbins Basic Pathology. 8th ed. Philadelphia PA: Elsevier; 2007. p. 878.
4. Wallace RJ Jr., Griffith DE. Antimycobacterial agents. In: Kasper DL, Fauci AS, Longo DL, Braunwald E, Hauser SL, Jameson JL, editors. Harrison's Principles of Internal Medicine. 16th ed. New York: McGraw-Hill; 2005. p. 946.
5. Petri WA Jr. Anti-microbial agents-drugs used in the chemotherapy of tuberculosis, *Mycobacterium avium* complex disease, and leprosy. In: Goodman Gilman's the Pharmacological Basis of Therapeutics. 10th ed. United States: McGraw-Hill Medical Publishing; 2001. p. 1273-82.
6. Grange JM, Zumla A. Manson's Tropical Diseases. 21st ed. Philadelphia, PA: Saunders; 2003. p. 1040.
7. McPhee SJ, Papadakis MA, Rabow MW. Current Medical Diagnosis and Treatment. 50th ed. United States: McGraw Hill Professional; 2011.
8. Steele MA, Burk RF, DesPrez RM. Toxic hepatitis with

- isoniazid and rifampin. A meta-analysis. *Chest* 1991;99:465-71.
9. Garibaldi RA, Drusin RE, Ferebee SH, Gregg MB. Isoniazid-associated hepatitis. Report of an outbreak. *Am Rev Respir Dis* 1972;106:357-65.
 10. Schaberg T, Rebhan K, Lode H. Risk factors for side-effects of isoniazid, rifampin and pyrazinamide in patients hospitalized for pulmonary tuberculosis. *Eur Respir J* 1996;9:2026-30.
 11. Devoto FM, González C, Iannantuono R, Serra HA, González CD, Sáenz C. Risk factors for hepatotoxicity induced by antituberculosis drugs. *Acta Physiol Pharmacol Ther Latinoam* 1997;47:197-202.
 12. Pande JN, Singh SP, Khilnani GC, Khilnani S, Tandon RK. Risk factors for hepatotoxicity from antituberculosis drugs: A case-control study. *Thorax* 1996;51:132-6.
 13. Sharma SK, Mohan A. In: Gupta SB, editor. *Antituberculosis Treatment Induced Hepatotoxicity: From Bench to Bedside*. Mumbai: Association of Physicians of India; 2005.

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