A Study of Changes in Liver Enzymes (ALT & AST) with Antitubercular Treatment in Newly Diagnosed Sputum Smear Positive Patients At Rims, Ranchi

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Abstract:
Introduction: Tuberculosis is caused by mycobacterium tuberculosis complex. India is the country with the highest burden of T.B.
Objective: To analyse changes in liver enzymes( ALT& AST) with anti tubercular treatment in newly diagnosed sputum smear positive pulmonary tuberculosis patient.
Methods: Data was obtained from O.P.D and Indoor patients of Department of Medicine., R.I.M.S, Ranchi who were suffering from pulmonary tuberculosis. 55 patients were included in this study of which 2 patients developed ATT induced hepatitis and 3 patients remained sputum smear positive even after completion of intensive phase and hence were excluded from the study.
Conclusion: There was an increase in liver enzyme level during intensive phase of antitubercular treatment while there was a decrease in liver enzyme level in continuation phase of treatment.
Keywords: Att, Liver Enzymes, Pulmonary Tuberculosis

I. Introduction

Tuberculosis which is caused by mycobacterium tuberculosis complex is one of the oldest disease known to affect humans and a major cause of death world wide.¹ M. tuberculosis is rod shaped non spore forming thin aerobic bacterium measuring 0.5µm by 3µm, an acid fast bacilli. It affect, lung intestine, meninges, bones, joints,lymphnodes,splendand other tissues of the body. India is the country with the highest burden of T.B. The WHO T.B statistics for India for 2015 give an estimated incidence figure of 2.2 million cases of T.B for India out of a global incidence of 9.6 million. In Jharkhand sputum smear positive patient diagnosed in 2015 were 20544.² The TB prevalence is the number of people in India who are living with active TB. Prevalence is usually, but not always given as a percentage of the population.³

Total treatment period for newly diagnosed tuberculosis patients is of 6 months including intensive and continuation phases (2 and 4 months, respectively). The intensive phase comprised of isoniazid INH (5 mg/kg day⁻¹; maximum 300 mg/day), rifampicin RIF (10 mg/kg day⁻¹; maximum 600 mg/day), ethambutol (EMB) (15–20 mg/kg day⁻¹), and pyrazinamide PZA (20–25 mg/kg day⁻¹). The continuation phase comprised daily similar doses of INH and RIF. Out of above drugs Pyrazinamide, rifampicin,isoniazid are hepatotoxic and cause increase in serum transaminase levels.

Thus present study is aimed to analyse changes in liver enzymes( ALT and AST) in different phases of treatment with antitubercular drugs.

II. Materials And Methods

Data was obtained from O.P.D and Indoor patients of Department of Medicine., R.I.M.S, Ranchi who were suffering from pulmonary tuberculosis.

Inclusion criteria:
1. Newly detected sputum smear positive pulmonary tuberculosis Patients

Exclusion Criteria:
1. HIV positive patients
2. History or any indication of hepatitis or cirrhosis
3. History of regular alcohol intake
4. Pregnancy or postpartum period
5. Abnormal baseline LFT
6. Patients remaining sputum smear positive after completion of 2 months of intensive phase of ATT
7. Patients with drug induced hepatitis due to ATT during course of treatment.
Diagnosis of ATT Induced Hepatitis:

Presence of at least one of the following:
1. A rise to more than 5 the upper limit of normal (ULN) level of ALT and/or AST
2. A rise in total serum bilirubin to more than 1.5 mg/dl
3. Any increase in AST and/or ALT above pre-treatment levels together with anorexia, nausea, vomiting, and jaundice.

Baseline Evaluation:
Baseline evaluation included clinical history, physical examination, sputum smear examination, chest radiograph, abdominal ultrasonography, complete blood cell count, ESR, LFT, and hepatitis markers.

Follow-up:
Patients were followed closely fortnightly over the first 2 months, then monthly till the end of the 6-month period. In each visit, patients were assessed clinically (response to therapy, any adverse effects, and nutritional status), and biochemically including LFTs, which were repeated whenever symptoms or signs suggestive of hepatotoxicity (nausea, anorexia, malaise, vomiting, hepatomegaly, or jaundice) occurred.

III. Results

Total 55 patients were included in this study of which 2 patients developed ATT induced hepatitis and 3 patients remained sputum smear positive even after completion of intensive phase and hence were excluded from the study.

<table>
<thead>
<tr>
<th>DURATION</th>
<th>MEAN± SD</th>
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<tbody>
<tr>
<td></td>
<td>ALT</td>
</tr>
<tr>
<td>Before initiation of therapy</td>
<td>19.28±4.7</td>
</tr>
<tr>
<td>1 month</td>
<td>60.44±34.2</td>
</tr>
<tr>
<td>2 month</td>
<td>64.10±31.8</td>
</tr>
<tr>
<td>3 month</td>
<td>51.32±14.9</td>
</tr>
<tr>
<td>4 month</td>
<td>47.28±11.0</td>
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<tr>
<td>5 month</td>
<td>40.38±7.5</td>
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<tr>
<td>6 month</td>
<td>37.60±7.8</td>
</tr>
</tbody>
</table>

Table No. 1

![Changes in Liver Enzymes in Patients Taking ATT](image-url)
IV. Discussion and Conclusion:

Baseline enzyme levels before therapy was within normal limits. Mean ALT level was 19.28±4.7 IU/ltr while Mean AST level was 20.20±5.6 IU/ltr. After 1 month of treatment most of the patients showed increase in liver enzymes, but were mostly asymptomatic with <3 fold elevation of serum transaminases. Mean ALT level at 2 month was 64.10±31.8 IU/ltr while Mean AST level was 48.42±12.0 IU/ltr. Thus at the end of intensive phase of treatment serum transaminase levels were in increasing trend. Mean ALT level at 3 month was 51.32±14.9 IU/ltr while Mean AST level was 40.60±8.8 IU/ltr. There was a decrease in transaminase level in continuation phase of treatment. This shows the additive effect of multidrug therapy.

Mean ALT level at the completion of continuation phase i.e at 6 month was 37.60±7.8 IU/ltr while Mean AST level was 32.46±5.9 IU/ltr. These values were further decreased from the earlier readings taken at intensive phase and reading after commencement of continuation phase. This result is consistent with previous studies done by K.C Chang, C.C Lang & C.M Tan. European Resp J 2007; De Souza AF, 1996; Singh J1996, Altman 1993.

Reference

[1]. Harrison Principle of Internal Medicine, 19th, 1102