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ANTI-TUBERCULAR TREATMENT INDUCED HEPATITIS

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ABSTRACT

Hepatotoxicity is a potentially serious adverse effect of isoniazid, rifampicin, and pyrazinamide-based anti-tubercular therapy regimens. Isoniazid (INH) induces asymptomatic transaminase elevations in approximately 10%-20% of individuals and hepatitis in approximately 1% of patients. After the emergence of jaundice, INH-induced hepatitis can be fatal in between 8% and 10% of individuals. An ATT regimen had been in place for the past two months for a 29-year-old female patient with a history of tuberculosis. For 45 days, she stopped taking her TB medicine, then resumed it for another 10 days before discontinuing it again. Constipation and pale skin began to show up on the patient's skin after a few days of treatment. It took only a few days of therapy before she began to show signs of malnutrition, constipation, and a pallid complexion. Scleral yellowing and vomiting were among the patient's later symptoms. Direct toxicity, idiosyncratic damage, the activation of liver enzymes, and allergic reactions all contribute to the pathophysiology of ATT-induced hepatotoxicity.

Key Words:- Hepatotoxicity, liver function, Tuberculosis, Adverse drug reaction

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INTRODUCTION

Hepatotoxicity is a potentially serious adverse effect of isoniazid, rifampicin, and pyrazinamide-based anti-tubercular therapy regimens. Isoniazid (INH) induces asymptomatic transaminase elevations in approximately 10%-20% of individuals and hepatitis in approximately 1% of patients [Gangadharan PRJ, 1986] [Gurumurlhy P, et. al., 1984]. After the emergence of jaundice, INH-induced hepatitis can be fatal in between 8% and 10% of individuals.

The mechanism by which INH causes hepatitis is unknown. Both direct toxicity and hypersensitivity have been implicated as possible pathogenic pathways. Rifampicin, a hepatotoxic medication, has been shown to exacerbate the hepatotoxicity of INH [RaIeigh JW, 1973] [Pilheu lA, et. al., 1981]. Individuals' acetylator status has been suggested to play a role in the development of hepatotoxicity. Different investigations have discovered that when isoniazid and rifampicin are administered simultaneously, both fast and slow acetylators are at an increased risk of getting drug-induced hepatitis [Mitchell JR, et. al., 1975] [Riska N, 1976]. On the other hand, another investigation discovered that acetylator status was irrelevant. Thus, the question of whether fast or slow acetylators of INH are more hepatotoxic remains unresolved. The purpose of this study was to determine the clinical and biochemical profile of individuals suffering hepatotoxicity as a result of anti-tubercular therapy (AIT) and to determine whether acetylator status is a significant predictor of INH-induced liver injury [Parasarlhy R,, et. al., 1986] [Brummer DL, 1971].

Case report:

An ATT regimen had been in place for the past two months for a 29-year-old female patient with a history of tuberculosis. For 45 days, she stopped taking her TB medicine, then resumed it for another 10 days before discontinuing it again. Then, after 45 days of yellowing of the sclera and two weeks of vomiting, she was admitted to the hospital. An Isoniazid-Rifampicin-Pyrazine combination was administered to her because

she had been previously diagnosed with tuberculosis (400 mg).

Constipation and pale skin began to show up on the patient's skin after a few days of treatment. It was only after she saw a worsening of symptoms, including yellowish staining of the sclera and vomiting, that she decided to discontinue using the ATT treatment medicines. At the time of the assessment, her vital signs were normal, but an analysis of her liver parameters revealed differences from the norm. Hepatitis B, hepatitis C, and HIV tests were all negative. Acid-fast bacilli testing was similarly negative, according to the results. Scan results showed no abnormalities in the abdomen or pelvic (bed side). A sub-segmental atelectasis was found in the left mid zone on a chest x-ray. These measurements were confirmed to be within normal limits.

TABLE 1: Medication chart

Name	Dose
Ondansetron	**4 mg**
Ursodeoxycholic acid	300 mg
L aspartate granules	3 gm
Lactulose	15 ml

The physician advised the following medications (Table 1) following the termination of anti-tubercular treatments. For the treatment of tuberculosis, other medications such as streptomycin, ethambutol, and levofloxacin were administered. Patient was found to have elevated levels of bilirubin (hyperbilirubinemia) and elevated hepatic enzymes due to ATT hepatotoxicity (SGOT, SGPT, ALP).

DISCUSSION:

A study found that 8-36 percent of Indians suffer from drug-induced liver damage. An increased risk of drug-induced hepatotoxicity is found in Asian countries because of the high prevalence of alcoholic and malnutrition-related diseases and the ethnic predisposition to hepatitis B and other infectious diseases.

During this time, the patient was taking Isoniazid, Rifampicin, and Pyrazinamide as part of an ATT regimen for two months.

It took only a few days of therapy before she began to show signs of malnutrition, constipation, and a pallid complexion. Scleral yellowing and vomiting were among the patient's later symptoms. Direct toxicity, idiosyncratic damage, the activation of liver enzymes, and allergic reactions all contribute to the pathophysiology of ATT-induced hepatotoxicity [Chest, 1985] [Black M, 1975].

Hepatotoxicity can be caused by the use of these three medications isoniazid, rifampicin, and pyrazinamide, which all have a significant risk for causing hepatotoxicity. There are people who cannot endure the adverse effects and discontinue taking anti-tubercular medications, reducing the treatment's effectiveness. These side effects can be caused by one, two or all three of the drugs. Hepatotoxicity with ATT medications is exacerbated by factors such as age, alcohol consumption, and pre-existing liver disease, as well as the use of other hepatotoxic drugs. This drug is three times more effective than Isoniazid or Rifampicin in inducing significant side effects, according to research.

CONCLUSION:

Our findings indicate that AIT-induced hepatotoxicity is prevalent and can be fatal. Along with INH and rifampicin, pyrazinamide may contribute to hepatotoxicity.

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