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DRUG-INDUCED (ATT) HEPATITIS WITH LEFT HYDROURETERONEPHROSIS AND COPD WITH SYNPNEUMONIC EFFUSION: A CASE REPORT

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ABSTRACT

Drug-induced hepatitis is a major adverse effect associated with antitubercular therapy (ATT), particularly when isoniazid, rifampicin, and pyrazinamide are used concurrently. This case report discusses a 59-year-old male diagnosed with pulmonary tuberculosis who developed drug- induced (ATT) hepatitis during therapy, along with left hydroureteronephrosis, chronic obstructive pulmonary disease (COPD), and synpneumonic effusion. The patient presented with fever, shortness of breath, dry cough, and jaundice. Laboratory investigations revealed elevated liver enzymes and bilirubin, while imaging showed bilateral renal calculi and pleural effusion. Treatment involved discontinuation of hepatotoxic drugs, initiation of supportive management, and close monitoring. The case highlights the importance of early detection and management of ATT-induced hepatotoxicity to prevent irreversible liver injury.

KEYWORDS: ATT-induced hepatitis, Hepatotoxicity, Tuberculosis, Hydroureteronephrosis, COPD, Pleural effusion.

INTRODUCTION

Standard anti-tubercular therapy (ATT) is crucial for the treatment of tuberculosis, which is still a major public health concern. However, it is limited by drug-induced liver impairment, which is a major cause of treatment stoppage and morbidity. Age, concomitant liver disease, and nutritional condition are host risk factors for ATT-induced hepatitis, which can vary from asymptomatic enzyme increase to severe hepatocellular damage.[1] Urinary flow obstruction, increasing dilatation of the collecting system, and perhaps loss of renal function if left untreated are the consequences of hydroureteronephrosis caused by ureteric calculi.[2] When pneumonia and pleural effusion are present, the management of chronic obstructive pulmonary disease (COPD) is made more difficult since these patients have increased respiratory morbidity and frequently need close monitoring and optimal medication. The difficulties of striking a balance between organ protection and efficient anti-tubercular and supportive treatment are demonstrated by this case of ATT-induced hepatitis with left hydroureteronephrosis, COPD, and pneumonia with mild pleural effusion, highlighting the critical role of clinical pharmacists in such complicated situations.[3]

CASE PRESENTATION

A 59-year-old male was admitted in hospital with complaints of fever since one month (intermittent with chills), shortness of breath on walking less than 100 meters, dry cough for one month, burning sensation in the chest, regurgitation of fluid into the mouth, and decreased appetite. The patient had a known history of pulmonary tuberculosis (K/C/O Pulmonary TB) and was on ATT regimen consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol.

PRESENT MEDICAL HISTORY

The patient presented with fever for one month (intermittent with chills), shortness of breath on walking less than 100 meters, dry cough for one month, burning sensation in the chest, regurgitation of fluid into the

mouth, and decreased appetite. He also noticed yellowish discoloration of eyes and urine for the past few days, suggestive of jaundice.

PAST MEDICAL HISTORY

It is a known case of pulmonary tuberculosis (K/C/O Pulmonary TB) and has been on anti- tubercular therapy (ATT) comprising isoniazid, rifampicin, pyrazinamide, and ethambutol. No known history of diabetes mellitus, hypertension, or cardiovascular disease.

PERSONAL HISTORY

The patient is a chronic smoker and alcoholic but discontinued both habits 15 days prior to admission. He follows a mixed diet, with decreased appetite noted recently. Sleep is adequate, and bowel and bladder habits are normal.

PHYSICAL EXAMINATION

On examination, the patient was conscious and oriented, afebrile, and icterus was present. There was no cyanosis, edema, or clubbing. The patient was of thin build, with mild tenderness noted on abdominal palpation. Jaundice was clinically evident.

VITAL SIGNS (Daily Monitoring)

Parameter	Day 1 to 3	Day 4 to 6	Day 7 to 8	Day 9
Temperature (°F)	98.7	96.7	96.9	96
Blood pressure (mmHg)	100/70	100/60	120/70	120/70
Pulse rate (bpm)	92	82	90	88
Respiratory Rate (cpm)	18	18	18	18

INVESTIGATIONS

Laboratory investigations revealed elevated liver enzymes and bilirubin levels indicating hepatic involvement. Urine examination showed presence of oxalate crystals and RBCs. Imaging studies confirmed bilateral renal calculi, left gross hydroureteronephrosis, right mild pleural effusion, and prostatomegaly.

TREATMENT CHART

Drug Name	Generic Name	Route/Dose	Frequency	Indication
Inj. PAN	Pantoprazole	IV / 40 mg	OD	Gastroprotection and relief of reflux symptoms (burning chest, regurgitation) and to prevent stress-related/NSAID-related upper GI irritation while on multiple drugs.
Inj. ZOFER	Ondansetron	IV / 4 mg	SOS	Symptomatic control of nausea and vomiting that may occur due to hepatitis, antibiotics, and other medications.
Neb. Duolin	Ipratropium bromide+ Levo salbutamol	NS/2.5 ml (1 nebule)	TID	Bronchodilation to relieve dyspnea in a patient with COPD and pneumonia, improving airflow and oxygenation.
Tab. UDILIV	Ursodeoxycholic acid	PO / 300 mg	BD	Hepatoprotective therapy to support bile flow and help manage cholestatic features of drug-induced hepatitis (jaundice, elevated bilirubin).
Inj. MONOCEF	Ceftriaxone	IV / 1 gm	BD	Empiric/targeted antibiotic for suspected or confirmed bacterial infection, likely pneumonia or urinary/renal infection related to hydroureteronephrosis and calculi.
Inj. STREPTOMYCIN	Streptomycin	IM / 0.75 gm	OD	Second-line anti-tubercular drug used as part of a modified ATT regimen after stopping hepatotoxic agents (e.g., INH/RIF/PZA) due to ATT-induced hepatitis.
Tab. ETHAMBUTOL	Ethambutol	PO / 800 mg	OD	Continuation of non-hepatotoxic component of ATT regimen in view of pulmonary tuberculosis when other hepatotoxic drugs are withheld.
Tab. MOXIFLOXACIN	Moxifloxacin	PO / 400 mg	OD	Fluoroquinolone added as a replacement/alternative anti-TB agent in a liver-sparing ATT regimen and also provides additional coverage against respiratory pathogens in pneumonia.

DISCUSSION

ATT-induced hepatotoxicity remains a significant clinical concern due to the combined use of hepatotoxic agents. In this patient, the concurrent elevation of serum bilirubin and transaminases, along with clinical jaundice, supports the diagnosis of drug-induced hepatitis. The presence of renal calculi and hydroureteronephrosis further complicated management, as dehydration and altered metabolism may exacerbate drug toxicity. COPD with syn pneumonic effusion likely worsened the respiratory distress. Therapeutic management included discontinuation of hepatotoxic ATT drugs, supportive care, antibiotics, and use of hepatoprotective agents like ursodeoxycholic acid.

CONCLUSION

This case emphasizes the need for close monitoring of liver function in patients receiving ATT, especially when multiple hepatotoxic agents are administered concurrently. Early recognition of symptoms and appropriate therapeutic interventions are crucial to prevent irreversible liver injury and optimize patient outcomes.

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