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Dual presentation of drug-induced gastritis and hepatotoxicity during first-line ATT: A case report

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Abstract
Background: Anti-tuberculosis therapy (ATT) is essential for treating pulmonary tuberculosis but is often associated with adverse drug reactions (ADRs). Hepatotoxicity and gastrointestinal (GI) intolerance are among the most frequent complications. Their concurrent occurrence, however, poses diagnostic and therapeutic challenges.
Case Presentation: A 60-year-old male with microbiologically confirmed pulmonary tuberculosis was started on first-line ATT. Within 10 days, he developed recurrent vomiting, abdominal pain, loss of appetite, giddiness, fatigue, and shortness of breath. On admission, he was conscious, oriented, afebrile, and hemodynamically stable. Abdominal examination revealed diffuse tenderness. Laboratory investigations showed marked hyperbilirubinemia (total bilirubin 10.6 mg/dL, direct bilirubin 6.6 mg/dL), elevated transaminases (AST 173 U/L, ALT 87 U/L), raised alkaline phosphatase (223 U/L), hypoalbuminemia (3.0 g/dL), and altered A/G ratio. These findings indicated drug-induced liver injury. The gastrointestinal symptoms, occurring soon after ATT intake, were consistent with drug-induced gastritis.
ATT was discontinued, and the patient was managed with intravenous pantoprazole and ondansetron, along with oral ursodeoxycholic acid and N-acetylcysteine. He showed progressive symptomatic and biochemical improvement after withdrawal of the offending drugs. Further management included close monitoring of liver function tests with plans for reintroduction of modified ATT once hepatic function normalized.
Conclusion: This case highlights the dual presentation of ATT-induced gastritis and hepatotoxicity, emphasizing the need for clinicians to remain vigilant when patients develop persistent gastrointestinal complaints during therapy. Such symptoms may represent early warning signs of liver injury. Early recognition, prompt drug withdrawal, and timely supportive management are critical to prevent severe complications and to enable safe continuation of tuberculosis treatment.

Keywords: Tuberculosis, anti-tuberculosis therapy, drug-induced liver injury, drug-induced gastritis, adverse drug reactions

Introduction

Tuberculosis is a contagious disease that can affect multiple organs, including the bones, kidneys, and intestines, but most commonly involves the lung parenchyma, leading to pulmonary tuberculosis. It is caused by Mycobacterium tuberculosis, a rod-shaped, purple-staining bacterium transmitted through respiratory droplets from infected individuals. Typical clinical manifestations include persistent cough with sputum (sometimes blood-streaked in severe cases), weight loss, reduced appetite, fever, and night sweats. In India, the prevalence of TB is estimated at 256 cases per 100,000 population. Research evidence indicates that nearly 80% of cases can be successfully cured with Directly Observed Therapy (DOT) [1]. However, adverse drug reactions (ADRs) such as hepatotoxicity, gastrointestinal disturbances, and neurological complications may occur, which can increase mortality and reduce the overall effectiveness of treatment [3].
Although hepatotoxicity and GI intolerance are each well recognised, their concurrent presentation (drug-induced gastritis together with drug-induced liver disease) is less commonly emphasized in the literature and can complicate diagnosis and management. Overlapping symptoms — for example, vomiting and abdominal pain — may be attributed to either gastric irritation or hepatic dysfunction (or both), potentially delaying identification of

severe liver injury and appropriate drug cessation. Clinical reports and series of ATT-related adverse events show that early onset of significant liver injury may occur within days to weeks of therapy initiation and can be associated with prominent GI symptom [2].

Here we report a case of a 60-year-old male who developed drug-induced gastritis presenting with recurrent vomiting and abdominal pain, together with drug-induced liver disease characterized by marked jaundice and biochemical liver injury shortly after initiation of ATT. This case highlights the importance of early recognition of dual-organ adverse effects, prompt withdrawal of offending agents, and supportive care to permit safe re-introduction or modification of anti-tuberculosis therapy.

Case Presentation

A 60-year-old male was admitted to the thoracic ward with complaints of recurrent vomiting following anti-tuberculosis therapy (ATT) tablet intake for the past 5 days. The vomiting was non-bloody, contained mucus, and was associated with generalized abdominal pain, loss of appetite, giddiness, fatigue, and shortness of breath.

The patient had been microbiologically confirmed with pulmonary tuberculosis and was started on first-line ATT 10 days prior to admission. Within a few days of therapy, he began developing intolerance to the drugs in the form of persistent vomiting and gastrointestinal discomfort.

On examination, the patient was conscious, oriented, and afebrile. His vital signs were stable: blood pressure 110/70 mmHg, pulse rate 80 beats per minute, and SpO_2 97% on room air. Abdominal examination revealed mild diffuse tenderness, without hepatosplenomegaly or ascites.

Laboratory investigations (Table 1) demonstrated deranged liver function with significant hyperbilirubinemia (total bilirubin 10.6 mg/dL, direct bilirubin 6.6 mg/dL, indirect bilirubin 4.0 mg/dL), elevated transaminases (AST 173 U/L, ALT 87 U/L), raised alkaline phosphatase (223 U/L), hypoalbuminemia (3.0 g/dL), and altered A/G ratio (0.71). Complete blood count showed leukocytosis (WBC 12.8 $\times 10^9$/L) with borderline low hematocrit (36.2%). Renal function and electrolytes were within normal limits.

Table 1: Laboratory Investigations

Parameter	Value	Normal Range	Unit
WBC (White Blood Cells)	12.8	4.0 - 11.0	$\times 10^9$/L
RBC (Red Blood Cells)	4.47	4.5 - 5.9	$\times 10^{12}$/L
HCT (Hematocrit)	36.2	40 - 50	%
MCV (Mean Corpuscular Volume)	81	80 - 96	fL
MCH (Mean Corpuscular Hemoglobin)	26.4	27 - 33	pg
MCHC (Mean Corpuscular Hemoglobin Concentration)	32.6	32 - 36	g/dL
RBS (Random Blood Sugar)	77	70 - 140	mg/dL
Urea	28	15 - 40	mg/dL
Creatinine	0.74	0.6 - 1.2	mg/dL
Total Bilirubin	10.6	0.3 - 1.2	mg/dL
Direct Bilirubin	6.6	0.0 - 0.3	mg/dL
Indirect Bilirubin	4	0.2 - 0.9	mg/dL
SGOT (AST)	173	5 - 40	U/L
SGPT (ALT)	87	7 - 56	U/L
Alkaline Phosphatase	223	44 - 147	U/L
Albumin	3	3.5 - 5.0	g/dL
Globulin	4.2	2.0 - 3.5	g/dL
A/G Ratio	0.71	1.0 - 2.2	—
Serum Sodium	136	135 - 145	mmol/L
Serum Potassium	3.7	3.5 - 5.0	mmol/L
Serum Chloride	105	98 - 107	mmol/L

Based on the temporal relationship with drug intake, clinical features, and biochemical evidence, a diagnosis of drug-induced gastritis and drug-induced liver disease secondary to ATT was made. ATT was discontinued immediately. The patient was managed with supportive measures including intravenous pantoprazole 40 mg twice daily, intravenous ondansetron 4 mg twice daily, oral ursodeoxycholic acid 200 mg, and oral N-acetylcysteine 600 mg three times daily.

The patient showed symptomatic improvement following cessation of ATT and initiation of supportive therapy. Further management was planned with close monitoring of liver function tests and consideration of reintroduction of ATT under modified regimens once liver function normalized.

Discussion

Anti-tuberculosis therapy (ATT) remains the cornerstone of treatment for pulmonary tuberculosis but is frequently complicated by adverse drug reactions (ADRs). Among these, hepatotoxicity and gastrointestinal (GI) intolerance are the most common and clinically significant. The present case illustrates a dual presentation of drug-induced gastritis and drug-induced liver disease, occurring within 10 days of initiating first-line ATT.

Hepatotoxicity is one of the most serious ADRs associated with ATT, attributed mainly to isoniazid, rifampicin, and pyrazinamide. The reported incidence of ATT-induced hepatotoxicity varies widely from 2% to 28% depending on the population and monitoring protocols [4]. A prospective Indian study found that up to 22% of patients on ATT developed hepatotoxicity, highlighting the need for vigilant monitoring in high-burden regions [5].

The pathogenesis of DILI in ATT is multifactorial, including direct hepatotoxic effects of pyrazinamide, toxic metabolite accumulation from isoniazid, and immune-mediated mechanisms [6]. Clinical manifestations range from asymptomatic elevations in liver enzymes to jaundice, fulminant hepatic failure, and death. In this case, marked

hyperbilirubinemia with elevated transaminases and alkaline phosphatase indicated a mixed hepatocellular-cholestatic injury, consistent with previous reports [7].

GI intolerance is another frequent adverse effect of ATT, presenting as nausea, vomiting, abdominal pain, and anorexia. Studies estimate that 30-40% of patients on ATT experience some degree of GI disturbance. Rifampicin and pyrazinamide have been particularly implicated in causing gastric irritation and intolerance [8].

Case reports from India also document ATT-induced gastritis requiring drug withdrawal and supportive therapy. For example, a 46-year-old male developed severe gastritis with nausea, vomiting, and abdominal pain within weeks of starting ATT, which resolved upon discontinuation of therapy [7]. This aligns with our case, where recurrent vomiting and abdominal pain coexisted with biochemical evidence of liver injury.

Management of ATT-induced DILI and gastritis requires immediate cessation of offending drugs and initiation of supportive care. Guidelines recommend close monitoring of liver function tests and gradual reintroduction of drugs in a step-wise manner once hepatic function normalizes [9]. In this case, the patient responded well to pantoprazole and ondansetron for gastritis, while ursodeoxycholic acid (UDCA) and N-acetylcysteine (NAC) were used as hepatoprotective measures, both of which have been reported as beneficial adjuncts in ATT-DILI [10].

The dual presentation of gastritis and liver injury emphasizes the importance of not dismissing GI symptoms during ATT as trivial, since they may herald or accompany hepatotoxicity. Older age, as in this patient, is a known risk factor for severe ADRs, necessitating stricter monitoring protocols.

Conclusion

This case highlights the occurrence of drug-induced gastritis and liver injury in a patient undergoing first-line anti-tuberculosis therapy. Gastrointestinal symptoms such as recurrent vomiting and abdominal pain, though common and often considered minor, may coexist with or precede hepatotoxicity. Early recognition of these adverse drug reactions, prompt withdrawal of offending agents, and timely initiation of supportive therapy are critical to prevent severe complications. Careful monitoring of liver function and gradual reintroduction of ATT under supervision are essential to ensure both patient safety and treatment success.

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Ethical approval

Ethical approval was not required for this case report as per institutional guidelines, since it describes an individual clinical case without experimental intervention.

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