RESEARCH PAPER

Case Report on Isoniazid Toxicity in a 46-Year-Old Male with Abdominal Cocoon Syndrome and Tuberculous Peritonitis.

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ABSTRACT
Isoniazid (INH), a cornerstone of anti-tubercular therapy (ATT), is well-tolerated but can cause significant toxicity, including hepatotoxicity and neurotoxicity, in a subset of patients. This case report describes a 46-year-old male with abdominal cocoon syndrome secondary to tuberculous peritonitis who developed isoniazid toxicity during the course of treatment. The report outlines the clinical presentation, diagnostic workup, and management of both the underlying condition and the drug-induced toxicity.
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INTRODUCTION

Tuberculous peritonitis, a manifestation of extrapulmonary tuberculosis, can lead to complications such as abdominal cocoon syndrome (ACS). Anti-tubercular therapy, including isoniazid, is the mainstay of treatment. However, INH is associated with hepatotoxicity and neurotoxicity, which can complicate therapy and necessitate prompt recognition and intervention. This case emphasizes the importance of monitoring for drug-induced toxicity in patients undergoing ATT, especially in complex presentations such as ACS.

Case Presentation

A 46 years old male patient working as a paramedical staff in a government hospital was admitted with the complaints of

- Intermittent abdominal pain and distension for 1 year.
- Weight loss of 10 kg and loss of appetite over 6 months.
- Low-grade fever and night sweats.
- Severe abdominal pain, nausea, bilious vomiting, and inability to pass stool or flatus for 48 hours.

Past Medical History:

- Pulmonary tuberculosis treated 7 years ago with a 6-month ATT regimen.

The patient was initiated on a 4-drug ATT regimen, including isoniazid (300 mg daily).

By the third week of therapy, he developed the following:

- **Jaundice**: Yellowish discoloration of the sclera and skin.
- **Fatigue and Weakness**.
- **Nausea and Poor Appetite**.
- **Paresthesia**: Tingling in hands and feet.

Physical Examination

1. **General Examination**:
   - **Appearance**: Cachectic, visibly pale, and icteric sclera.
   - **Vitals**:
     - Blood pressure: 110/70 mmHg.
     - Pulse: 100 bpm, regular.
     - Respiratory rate: 18/min.
     - Temperature: 38.2°C.
   - No palpable lymphadenopathy or edema.
2. **Abdominal Examination**:
   - Diffuse abdominal distension with a firm and tense abdomen.
   - Mild tenderness without guarding or rebound tenderness.
   - Fluid thrill positive, indicating ascites.
   - Hyperactive bowel sounds.
3. **Neurological Examination**:
   - Reduced sensation in a glove-and-stocking distribution.

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- Reflexes preserved but slightly diminished in the lower limbs.
- Normal motor strength.

Laboratory Investigations
Baseline Investigations (Prior to ATT Initiation)

1. **Hematological Profile**:
   - Hemoglobin: 11.5 g/dL (mild anemia).
   - Total leukocyte count: 9,200/µL (within normal limits).
   - Platelet count: 280,000/µL.
2. **Liver and Renal Function Tests**:
   - ALT: 32 U/L, AST: 30 U/L (normal).
   - Total bilirubin: 0.8 mg/dL.
   - Serum creatinine: 0.9 mg/dL.
3. **Infectious Workup**:
   - HIV: Negative.
   - Mantoux test: Positive (induration 15 mm).
   - Ascitic fluid ADA: 56 U/L (indicative of tuberculosis).

During Isoniazid Toxicity

1. **Liver Function Tests**:
   - ALT: 160 U/L (5× upper limit of normal).
   - AST: 190 U/L.
   - Total bilirubin: 3.5 mg/dL (direct bilirubin: 2.2 mg/dL).
   - Elevated alkaline phosphatase: 140 U/L.
2. **Peripheral Blood Smear**:
   - No evidence of hemolysis.
3. **Serum Vitamin B6 (Pyridoxine)**:
   - Deficiency noted: 4.0 ng/mL (reference range >5 ng/mL).
4. **Coagulation Profile**:
   - Prothrombin time (PT): 16 seconds (slightly prolonged).
   - INR: 1.4.
5. **Complete Blood Count (CBC)**:
   - WBC: 11,500/µL (mild leukocytosis).
   - Hemoglobin: 10.2 g/dL.
   - Platelets: 320,000/µL.
6. **Ascitic Fluid Analysis**:
   - Exudative fluid: Protein 3.5 g/dL, Glucose 50 mg/dL.
   - Lymphocytic predominance (80%).
   - Negative for malignant cells.

Radiological Findings
1. Ultrasound Abdomen

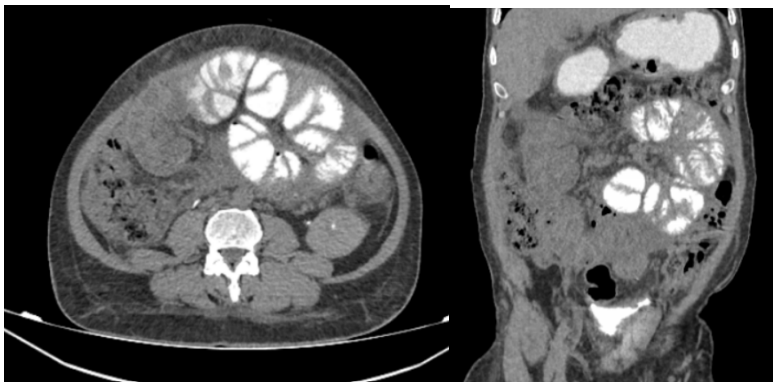
- Moderate ascites with internal septations.
- Thickened peritoneum with echogenic strands.
- Clustering of small bowel loops with limited peristalsis.

2. Contrast-Enhanced CT (CECT) Abdomen

- **Findings Consistent with Abdominal Cocoon Syndrome**:
  - Encapsulation of small bowel loops by a thick, fibrous membrane.
  - Enhancing peritoneal thickening and omental nodularity.
  - Moderate ascites with fine septations.
- **No evidence** of bowel ischemia or perforation.

3. Chest X-Ray

- Right upper lobe fibrotic changes, consistent with healed pulmonary tuberculosis.
- No active pulmonary lesions.



Diagnosis

1. Abdominal Cocoon Syndrome secondary to tuberculous peritonitis.
2. Drug-induced hepatotoxicity and peripheral neuropathy due to isoniazid.

Management

1. Surgical Management of Abdominal Cocoon Syndrome

- **Intraoperative Findings**:
  - Thick fibrous membrane encasing the small bowel.
  - Moderate ascites with thickened peritoneum and granulomatous nodules.
  - Viable bowel loops with no ischemia or perforation.
- **Procedure**:
  - Complete excision of the fibrous membrane.
  - Adhesiolysis to release small bowel loops.
  - Biopsies of the peritoneum and omental nodules.

2. Management of Isoniazid Toxicity

Hepatotoxicity:

- INH was immediately stopped upon detecting elevated liver enzymes and jaundice.
- The patient was transitioned to a modified ATT regimen:
  - **Rifampin, Ethambutol, Pyrazinamide**.
  - Streptomycin added as a fourth drug.
- Supportive care:
  - High-calorie, protein-rich diet.
  - Hepatoprotective agents (e.g., N-acetylcysteine).
  - Close monitoring of liver function tests biweekly.

Neurotoxicity:

- Supplementation with **Pyridoxine (Vitamin B6)** at 100 mg daily.
- Neurological symptoms gradually resolved within 2 weeks.

Postoperative Course

- **Recovery**: The patient had an uneventful postoperative course.
- Oral feeding was resumed by postoperative day 5.
- Liver enzymes normalized over 4 weeks after discontinuation of INH.
- The patient completed a 9-month ATT regimen with the modified drug regimen.
- **Histopathology**: Peritoneal biopsy confirmed chronic granulomatous inflammation with caseation, consistent with tuberculosis.

DISCUSSION

Isoniazid Toxicity

1. **Hepatotoxicity**:
   - Incidence: Affects 10–20% of patients on INH, with clinical hepatitis in 1–3%.
   - Mechanism: Formation of toxic metabolites during INH metabolism via the cytochrome P450 system.
   - Risk Factors: Older age, alcohol use, pre-existing liver disease, and genetic predisposition (slow acetylators).
2. **Neurotoxicity**:
   - Mechanism: INH-induced depletion of pyridoxine, leading to impaired synthesis of neurotransmitters.
   - Manifestations: Peripheral neuropathy, ataxia, or seizures in severe cases.

Abdominal Cocoon Syndrome and Tuberculous Peritonitis

- Tuberculous peritonitis is a common cause of secondary ACS in endemic regions.
- Chronic granulomatous inflammation and peritoneal fibrosis lead to bowel encapsulation.
- Surgical excision of the fibrous membrane is curative in most cases.

Challenges in Management

- **Overlap of Symptoms**: Hepatotoxicity symptoms (e.g., abdominal pain and jaundice) may mimic TB progression or complications of ACS.
- **Modified ATT**: Requires careful selection of drugs to maintain efficacy while avoiding further toxicity.

CONCLUSION

This case illustrates the complexity of managing abdominal cocoon syndrome secondary to tuberculous peritonitis, compounded by isoniazid-induced hepatotoxicity and neurotoxicity. Early recognition of drug toxicity, prompt modification of ATT, and effective surgical intervention ensured a favorable outcome. Clinicians must remain vigilant for adverse drug reactions during ATT, especially in patients with comorbidities.

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