Research Article

Integrative Management of Adverse Drug Reactions in Pulmonary Tuberculosis: An Ayurvedic Case Report and Phytochemical Analysis

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Abstract

The efficient management of pulmonary tuberculosis (TB) is mainly contingent on strict patient compliance with first-line Anti-Tubercular Treatment (ATT). [1,2] Nonetheless, adverse drug reactions (ADRs)—spanning hepatotoxicity and peripheral neuropathy to considerable gastrointestinal distress and quick weight reduction—often result in treatment withdrawal, culminating in multi-drug-resistant TB (MDR-TB). [3,6] In Ayurveda, the pathophysiology of tuberculosis corresponds to Rajayakshma ("King of Diseases") and Kshaya (gradual tissue degeneration). [4] Conventional Ayurvedic medicines aim to restore digestive fire (Agni), eradicate channel blockages (Srotorodha), and cure systemic tissue deterioration (Dhatukshaya). [5] A 30-year-old lady reported severe involuntary weight loss (decreasing from 41 kg to 33 kg), shortness of breath, and continuing thoracic discomfort. After a chest X-ray revealed the existence of active pulmonary tuberculosis, traditional antitubercular medication was initiated. The patient then had incapacitating adverse medication reactions, such as chronic nausea, hair loss, muscle spasms, and acute tiredness, which remained after conventional treatment changes.

Interventions: An integrative, phase-based Ayurvedic adjunctive program was designed with ATT. Phase 1 covered the administration of Sitopaladi Churna and Mahamrugank Rasa combined with goat milk (Aja Dugdha). Phase 2 amalgamated Mahalaxmi Vilas with a composite herbal composition including Abhrak Bhasma, Srunga Churna, Pushkarmool (Inula racemosa), and Sitopaladi Churna blended with honey.

Results: During a four-month timeframe, the patient displayed alleviation of ATT-related gastrointestinal toxicity, stability and reversal of weight reduction, relief from neuromuscular spasms, and considerable enhancement in respiratory metrics.

Conclusion: Ayurvedic adjuvants—specifically Sitopaladi Churna combined with biocompatible vehicles—mitigate ATT toxicity through antioxidant, hepatoprotective, and bioenhancing pathways, presenting a feasible option for maintaining uninterrupted antitubercular treatment.

Keywords: Pulmonary Tuberculosis, Adverse Drug Reactions (ADRs), Anti-Tubercular Treatment (ATT), Ayurveda, Rajayakshma, Integrative Medicine, Sitopaladi Churna, Phytochemical Analysis, Bioenhancer (Piperine), Antitubercular Drug-Induced Liver Injury (ATDILI).

1. Introduction

Tuberculosis (TB) is one of the leading infectious causes of mortality globally, with Mycobacterium tuberculosis infecting millions annually [1, 36]. Global efforts, especially India's Pradhan Mantri TB Mukt Bharat Abhiyaan, prioritise early diagnosis and continuous multi-drug anti-tubercular treatment (ATT) regimens consisting of Isoniazid (H), Rifampicin (R), Pyrazinamide (Z), and Ethambutol (E) [2]. Despite great performance, conventional chemotherapy is limited by a broad spectrum of adverse drug reactions (ADRs) [3,15].

The clinical incidence of ATT-induced toxicity ranges from non-life-threatening gastrointestinal distress (nausea, anorexia, vomiting) to severe organ failure, including antitubercular drug-induced liver injury (ATDILI) and peripheral neurotoxicity [6]. These unfavourable consequences destabilise therapy, prompting deliberate medication default, sub-therapeutic dosage, and drug resistance [3,7].

To overcome compliance issues, interest has shifted toward integrative medicine [5]. In Ayurveda, TB-like wasting disorders have been categorised for millennia under Rajayakshma [4,8]. Ayurvedic co-therapies have dual utility: they protect organ systems from drug toxicity while modifying metabolic pathways to increase antibiotic bioefficacy [5,9].

2. Pathophysiological Correlation: Rajayakshma, Kshaya, and Tuberculosis

2.1 Ancient Etymology and Philosophical Basis

In classical Ayurvedic literature, Tuberculosis is regarded as Rajayakshma—literally translated as the "King" (Raja) of "Diseases or Decay" (Yakshma). It is closely synonymous with Kshaya (diminution/wasting of body tissues) and Shosha (emaciation or drying up of critical body fluids) [4]. Classical treatises, such as the Charaka Samhita [10] and Susruta Samhita [11], categorise Rajayakshma not just as a localised pulmonary illness, but as a systemic, multi-organ metabolic syndrome.

- **Etiological Factors (Nidana):** Sahasaja, Vegadharana, Kshayaja, Vishamashana.
- **Pathological Cascade:** Impairment of Digestive Fire (Agnimandya) → Accumulation of Undigested Toxins (Ama) & Channel Blockage (Srotorodha) → Tridosha Vitiation → Sequential Tissue Starvation & Wasting (Dhatukshaya) [Rasa → Rakta → Mamsa → Meda → Asthi → Majja → Shukra] → Depletion of Immune Reserve (Ojokshaya) → Opportunistic Pulmonary Infection & Systemic Collapse (Rajayakshma) [12,13,14].

2.2 Etiology (Nidana)

The Charaka Samhita defines four primary etiological vectors (Nidana-Chatustaya) triggering Rajayakshma [10]:

- **Sahasaja (Physical Overexertion):** Engaging in physical exertion beyond individual physiological capacity, causing internal pulmonic micro-trauma (Urakshata).
- **Vegadharana (Suppression of Natural Urges):** Habitual suppression of physical reflexes, leading to retrograde movement of Vata (Pratiloma Vata).
- **Kshayaja (Nutritional and Vital Depletion):** Progressive loss of vital tissue elements secondary to chronic starvation, emotional grief, or excessive physical depletion.
- **Vishamashana (Irregular Dietary Habits):** Consuming biologically incompatible, deficient, or improperly timed food, directly inducing metabolic dysfunction.

2.3 Pathogenesis (Samprapti) and Tissue Wasting (Dhatukshaya)

The fundamental pathogenic lesion in Rajayakshma is Agnimandya (derangement of central digestive and cellular metabolic fire). When Agni fails, food is incorrectly digested into Ama (toxic metabolic intermediate). Ama mixes with vitiated Kapha to generate Srotorodha—the physical and functional blocking of the nutrient-transport channels (Rasavaha Srotas) [14]. This blockage leads to:

- **Anuloma Kshaya:** Tissue starvation progressing sequentially from the primary nutrient fluid down to the reproductive tissue.
- **Pratiloma Kshaya:** Tissue loss initiated at deeper layers that retrogradely drains the body's foundational vitality (Ojas).

As Dhatukshaya advances, the host experiences Ojokshaya (profound immunodeficiency) [13]. This compromised state creates the permissive cellular environment necessary for Mycobacterium tuberculosis to colonise pulmonary tissue, establish granulomatous lesions, and induce systemic wasting.

2.4 Correlation of Clinical Manifestations (Ekadasha-Rupa)

Ayurvedic texts describe eleven cardinal symptoms (Ekadasha-Rupa) that characterise fully developed Rajayakshma, each matching features of pulmonary TB and ATT toxicity:

Classical Symptom (Ekadasha-Rupa)	Modern Clinical Correlation	Pathophysiological Mechanism
Shira Shula	Chronic Headache	Vata vitiation & ATT neurotoxicity
Kasa	Persistent Cough	Airway inflammation & alveolar necrosis
Shwasa / Galaddhvamsa	Dyspnea & Throat Irritation	Upper and lower respiratory mucosal ulceration
Svarabheda	Hoarseness of Voice	Laryngeal TB / mucosal drying (Shosha)
Jvara	Diurnal / Low-Grade Fever	Pyrexia of infectious origin & cytokine surge
Sashonita Sthivana	Hemoptysis	Structural erosion of pulmonary vasculature
Parshvashula	Pleuritic Chest Pain	Intercostal inflammation & pleural involvement
Arochaka	Anorexia & Nausea	ATT-induced GI irritation & Agnimandya

Atisara / Varchobheda	Diarrhea / Malabsorption	Intestinal dysbiosis & Rasavaha Srotorodha
Amsatapana	Burning Sensation in Shoulders	Systemic inflammation & metabolic toxicity
Dhatukshaya / Loss of Weight	Progressive Wasting / Cachexia	Hypermetabolic catabolism & Dhatu decay

3. Case Presentation

3.1 Patient Demographics and Baseline Presentation

A 30-year-old married female healthcare worker residing in Karad, Maharashtra, India, presented with a 12-month history of gradual physical deterioration. She experienced generalised asthenia, persisting low-grade evening fevers, chronic sore throat, exertion-induced dyspnea, and regional pleuritic chest discomfort. Over the prior year, her body mass dropped from a baseline of 41 kg to 33 kg (BMI: 13.7 kg/m2, indicating severe adult malnutrition).

3.2 Diagnostic Assessment and Initiation of First-Line ATT

On October 26, 2024, diagnostic evaluation at a regional government hospital confirmed pulmonary tuberculosis via sputum smear positivity and a chest X-ray demonstrating focal consolidations with mid-zone cavitation. She was immediately started on standard first-line weight-banded ATT:

- Isoniazid (H): 300 mg/day
- Rifampicin (R): 450 mg/day
- Pyrazinamide (Z): 1000 mg/day
- Ethambutol (E): 800 mg/day

3.3 Adverse Drug Reaction (ADR) Profile

Within three weeks of ATT initiation, the patient developed severe adverse drug reactions:

- **Gastrointestinal:** Severe, persistent nausea, complete loss of appetite, epigastric distress, and frequent bilious vomiting.
- **Neuromuscular:** Intense nocturnal foot cramps, distal extremity paresthesias, severe bilateral temporal headaches, and muscle weakness.
- **Dermatological/Integumentary:** Diffuse hair shedding (alopecia) and xerosis.
- **Anthropometric:** Continued, unchecked weight loss down to 33 kg.

On November 24, 2024, normal allopathic symptomatic therapy (antiemetics, proton-pump inhibitors, and pyridoxine supplements) was commenced, and ATT dose was modified, but her symptoms increased throughout the following month. Fearing lasting organ damage and absolute intolerance to chemotherapy, she sought supplementary Ayurvedic intervention.

4. Therapeutic Intervention and Treatment Protocol

The therapeutic strategy attempted to manage ATT toxicity without compromising antitubercular efficacy. The Ayurvedic strategy centred on Deepana (kindling metabolic fire), Pachana (digesting metabolic poisons), Srotoshodhana (clearing channel blockages), and Brimhana/Rasayana (restoring tissue mass and cellular immunity) [8,30].

4.1 Phase 1 Regimen (Months 1–2): Acute Toxicity Mitigation

- **Sitopaladi Churna:** 5 g twice daily (BD) post-meals.
  - Anupana (Vehicle): 100 mL warmed Aja Dugdha (Goat milk).
- **Mahamrugank Rasa:** 100 mg twice daily (BD).
  - Anupana: Warmed water / Honey.

4.2 Phase 2 Regimen (Months 3–4): Deep Tissue Rejuvenation

- **Mahalaxmi Vilas Rasa:** 125 mg twice daily (BD) with honey.
- **Compounded Respiratory & Anabolic Churna:** Administered twice daily with honey, containing:
  - Abhrak Bhasma (Shataputi): 125 mg

- Pushkarmool Churna (Inula racemosa): 250 mg
- Srunga Bhasma (Pistacia integerrima): 250 mg
- Sitopaladi Churna: 500 mg

5. Phytochemical, Pharmacodynamic, and Pharmacokinetic Mechanisms of Sitopaladi Churna

Constituents of Sitopaladi Churna		
Pippali (Piper longum)	Twak (Cinnamomum)	Ela (Elettaria)
[Piperine Bioenhancer]	[Eugenol & Cinnamaldehyde]	
P-gp & CYP3A4 Inhibition	Free Radical Scavenging	Glutathione Restoration
↑ Rifampicin Bioavailability (Elevated Cmax & AUC)	↓ ATDILI / Hepatotoxicity (INH/PZA Protection)	↓ Mucosal Inflammation (Anti-emetic Effect)

5.1 Composition and Active Secondary Metabolites

Sitopaladi Churna is a classical polyherbal formulation composed of five ingredients [16,17]:

Ingredient	**Botanical / Common Name**	**Ratio**	**Primary Bioactive Secondary Metabolites**
Sitopala	Saccharum officinarum (Sugar Candy)	16 Parts	Pure Sucrose, Disaccharides (Demulcent Base)
Vamshalochana	Secretion of Bambusa bambos (Bamboo Manna)	8 Parts	Amorphous Silica (SiO_2), Calcium, Silicates
Pippali	Piper longum (Long Pepper)	4 Parts	Piperine, Piperlongumine, Piperatines
Ela	Elettaria cardamomum (Cardamom)	2 Parts	1,8-Cineole, α-Terpinyl Acetate, Sabinene
Twak	Cinnamomum zeylanicum (Cinnamon)	1 Part	Cinnamaldehyde, Eugenol, Trans-Cinnamic Acid

5.2 Modulation of Bioavailability via Piperine

Piperine, the principal alkaloid contained in Pippali, serves as a potent, non-toxic bioenhancer (Yogavahi) [18].

- **P-Glycoprotein Inhibition:** Piperine quickly binds to and obstructs ATP-dependent P-glycoprotein transporters on intestinal enterocytes, improving mucosal absorption of Rifampicin [19,20].
- **CYP3A4 and UGT Inhibition:** Piperine non-competitively obstructs hepatic CYP3A4 and phase II UDP-glucuronosyltransferase (UGT), slowing hepatic clearance and increasing peak plasma concentration (C_{max}) and $AUC_{0-\infty}$ without generating hazardous drug accumulation [21].
- **Epithelial Permeability:** It modifies the lipid milieu of the intestinal brush-border membrane to improve drug diffusion [22].

5.3 Hepatoprotective and Anti-Oxidative Mechanisms

Antitubercular Drug-Induced Liver Injury (ATDILI) is largely linked to INH and PZA [6]. Sitopaladi Churna protects against this via:

- **Eugenol and Cinnamaldehyde (Twak):** Potent free radical scavengers that inhibit lipid peroxidation and upregulate Nrf2 to boost downstream production of endogenous antioxidant enzymes (SOD, CAT, GPx) [23,24].
- **1,8-Cineole (Ela):** Prevents depletion of intracellular reduced glutathione (GSH) in liver tissue produced by PZA metabolites [25].
- **Silica Matrix (Vamshalochana):** Adsorbs metabolic toxins and lowers hepatic inflammatory signals.

5.4 Gastrointestinal Mucosal Defence

Pippali and Twak downregulate local stomach pro-inflammatory cytokines. The demulcent feature of Sitopala produces a protective physical layer over the gastric epithelium, limiting direct acid-parietal interactions, while 1,8-cineole promotes stomach emptying and demonstrates anti-spasmodic actions [25].

5.5 Immunomodulatory Actions

Phytochemicals in Pippali limit the generation of histamine and leukotrienes from sensitised lung mast cells, regulating hypersensitivity reactions [26]. By maintaining the intestinal microbiota from severe antibiotic dysbiosis, Sitopaladi Churna maintains gut-lung axis communication.

6. Pharmacological Rationale of Co-Formulations and Vehicle (Anupana)

6.1 Aja Dugdha (Goat Milk)

In classical Ayurveda, Aja Dugdha (Goat milk) is specifically prescribed for Kshaya and Rajayakshma. Nutritional analysis indicates its clinical efficacy over cow's milk in cachectic patients:

Fat Globule Architecture: Goat milk fat globules average < 3.5 µm in diameter (compared to> 4.5 µm in cow's milk), affording a broader surface area for pancreatic lipase breakdown and enabling rapid digestion despite weak digestive fire (Agnimandya).

Medium-Chain Triglycerides (MCTs): High amount of caproic, caprylic, and capric acids 30–35% in goat milk vs. 15–20% in cow milk). MCTs are rapidly absorbed into the portal circulation without requiring bile acid emulsification, providing a rapid energy supply for critically malnourished people.

Micronutrient Bioavailability: Higher levels of bioavailable calcium, phosphorus, magnesium, and iron promote bone density and expedite cellular recovery.

Goat Milk (Aja Dugdha)	**Cow Milk (Go Dugdha)**
Fat Globule Size: <3.5 µm	Fat Globule Size: >4.5 µm
MCT Content: 30–35%	MCT Content: 15–20%
Direct Portal Absorption (No Bile)	Requires Biliary Emulsification
Rapid Assimilation in Agnimandya	Slower Digestion / Potential Stasis

Goat milk affords a broader surface area for pancreatic lipase breakdown and possesses high Medium-Chain Triglycerides (MCTs) for rapid energy supply in malnourished patients [27,28,29].

6.2 Abhrak Bhasma and Pushkarmool

- **Abhrak Bhasma:** Standardised iron-silicate nanoparticles (20-100 nm) that bypass hepatic first-pass degradation and cross cellular membranes to deliver trace elements and upregulate ATP synthesis in hypoxic tissues [32,33].
- **Pushkarmool (Inula racemosa):** Includes active sesquiterpene lactones that inhibit cyclic nucleotide phosphodiesterase (PDE) for bronchodilation, and suppress the NF-κB signalling pathway to mitigate pleuritic chest pain [34,35].

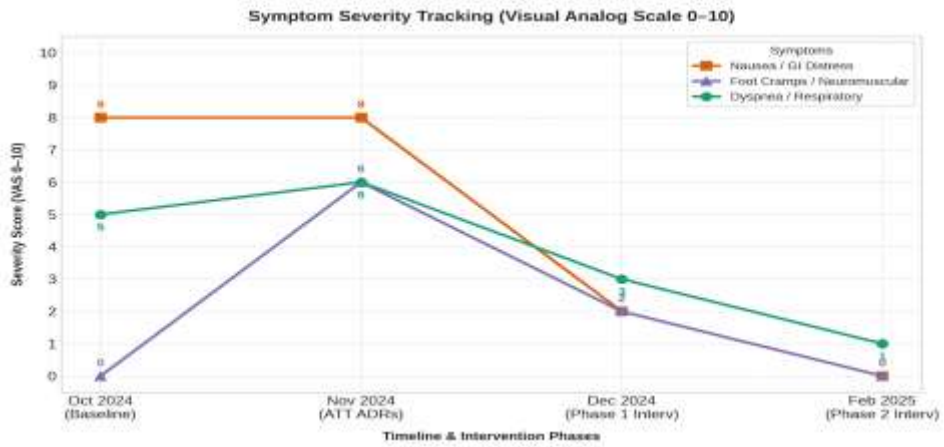
7. Clinical Outcomes and Discussion

7.1 Quantitative Symptom Progression

Over the 16-week intervention period, the patient experienced progressive, measurable improvement across all primary complaint domains.

Metric / Symptom	**Pre-Intervention**	**Post-Intervention**
Body Mass	33 kg (Severe Cachexia)	39.5 kg (Progressive Gain)
Nausea / GI Distress	Continuous (VAS 8/10)	Resolved (VAS 0/10)
Neuromuscular Cramps	Severe Nocturnal (VAS 6/10)	Resolved (VAS 0/10)
Exertional Dyspnea	Moderate (VAS 5/10)	Mild/Minimal (VAS 1/10)
ATT Compliance	Threatened (At Risk)	100% Adherence Achieved

Nausea and epigastric burning resolved within 14 days of Phase 1 initiation, allowing full dietary intake. Weight loss halted within 3 weeks, yielding a total gain of 6.5 kg over 4 months. Paresthesias, nocturnal cramps, and dyspnea decreased substantially due to the protocol's integrated metabolic and bronchodilatory support.



7.2 Integrative Medical Management Context

Standard allopathic management depends on symptom-targeted medicines, which increase polypharmacy and hepatotoxic burden. This case suggests an integrative approach protects target organs from drug-induced injury, increases bioavailability, and restores host physiological capability without affecting the bactericidal activity of antibiotics [5].

8. Conclusion

Antitubercular therapy default driven by drug toxicity is a fundamental vulnerability in worldwide tuberculosis control. This instance demonstrates how old Ayurvedic notions of Rajayakshma and Kshaya can be translated into successful supplementary therapy. The administration of Sitopaladi Churna, Abhrak Bhasma, Pushkarmool, and Aja Dugdha alongside standard chemotherapy abolished ATT-induced gastrointestinal toxicity, averted liver injury, recovered body mass, and obtained 100% compliance. Integrative regimens combining conventional bactericidal antibiotics with Ayurvedic supporting medicines require further research through official clinical studies.

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