

“Ethambutol-Associated Lichenoid Drug Eruption: A Case Report Emphasizing Adr Monitoring”

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ABSTRACT:

Tuberculosis is a highly contagious bacterial infection and known to be one of the leading causes of mortality worldwide. A combination of antibiotics used for 6 months, mainly first line therapy including isoniazid, rifampicin, pyrazinamide, and ethambutol is essential for management, at the same time it is also associated with various ADR's including severe cutaneous manifestations. Managing these ADR's poses significant challenges, as discontinuation of antitubercular therapy may increase the risk of disease progression and multidrug resistance. Drug-induced lichenoid photosensitivity is an uncommon yet clinically significant adverse reaction. Proper identification and diagnosis plays an important role in not only minimizing complications but also to reduce disease progression by uninterrupted the ATT. This case highlights the importance of identifying and effectively managing drug-induced lichenoid eruptions associated with antitubercular therapy without interrupting the course of antitubercular therapy. We describe a 64-year-old female diagnosed with active pulmonary tuberculosis and developed rare cutaneous adverse reaction during the course of ATT. The case underscores the need of early detection, diagnosis, treatment strategies ensuring continuation of ATT regimen and the vital role of pharmacovigilance in promoting patient safety.

Key Words: Ethambutol, Antitubercular therapy, Cutaneous drug eruption, Adverse Drug Reactions.

I. INTRODUCTION:

India bears the largest proportion of tuberculosis cases characterizing it as public health concern. Anti-tubercular therapy mainly first-line drugs plays a crucial role in the management of an airborne infectious disease [1]. The most common adverse effects of anti-tubercular drugs are gastrointestinal and hepatotoxic effects. Cutaneous Adverse Drug Reactions (CADR) of anti-tubercular

drugs like maculopapular rashes, purpuric skin eruptions, exfoliative dermatitis, toxic epidermal necrolysis and lichenoid drug eruptions are very uncommon [2]. Lichenoid drug eruption resembles with clinical features lichen planus which can cause symptomatic rash to life-threatening condition like flat-topped, violaceous to hyper pigmented, and itchy papules and plaques, distributed over the trunk and extremities and appearing after a period of few weeks to months after initiating the offending drug. This period between initiation of drug intake and eruption of lesions is known as the latent period [3]. This report aims to highlight the case of a 64-year-old female with a known case of Pulmonary TB who developed drug-induced lichenoid Eruptions during treatment with anti-tubercular drugs at Ballari Institute of medical sciences, Karnataka, India.

II. CASE REPORT:

A 64-year-old female patient was admitted to BMC-RC (A tertiary care teaching hospital), Ballari, Karnataka, India with a known diagnosis of active pulmonary tuberculosis. She had been receiving a standard first-line Anti-Tubercular Therapy (ATT) regimen comprising Isoniazid, Rifampicin, and Ethambutol for the preceding four months.

She presented with complaints of intermittent high-grade fever accompanied by chills, dry, non-productive cough, and breathlessness, with orthopnea noted over the last three days. During the course of therapy, she gradually developed diffuse, pruritic violaceous scaly eruptions over the body, associated with generalized hyper pigmentation, raising clinical suspicion for a lichenoid drug reaction.

The patient is a known case of pulmonary tuberculosis initially diagnosed in 2022. She was commenced on anti-tubercular therapy and continued treatment for approximately four months. However, during the course of therapy, she

developed ATT induced lichenoid eruptions, leading to treatment interruption and initiation of dermatological management. She underwent photosensitivity therapy twice weekly for one month as part of symptomatic control and skin lesion recovery.

Approximately five months ago, the patient experienced a recurrence of pulmonary tuberculosis and was reintroduced to standard anti tubercular therapy consisting of Isoniazide, Rifampicin, and Ethambutol. Despite the clinical necessity, the patient once again developed lichenoid eruptions within one week of restarting the regimen, suggestive of a recurrent drug induced dermatological hypersensitivity reaction.

General Appearance: Revealed widespread hyper pigmented, violaceous papules and plaques with coarse scaling, predominantly involving the trunk and extremities. Pedal edema was present.

ON CLINICAL EXAMINATION:

Vital Signs: Within physiological limits

Cardiovascular System: Normal S1 and S2, no audible murmurs

Central Nervous System: Fully conscious and well-oriented, without focal deficits

Respiratory System: Bilateral normal vesicular breath sounds, no added sounds

Abdomen: Soft, non-tender, no hepatosplenomegaly

Drug History:

- Isoniazid
- Rifampicin
- Ethambutol

Based on the chronological association, dermatological morphology, and clinical evolution, the eruption was strongly attributed to Ethambutol, thereby diagnosing an Ethambutol-induced Lichenoid Drug Eruption.

LABORATORY INVESTIGATION:

	Parameters	Result	Reference Range
COMPLETE BLOOD COUNT	Haemoglobin	10.9	12.5-16g
	Total WBC count	3490	4000-11000cells/cumm
	Neutrophils	48	40-70%
	Lymphocytes	42	20-40%
	Eosinophils	04	3-6%
	Monocytes	06	2-10%
	Basophils	0	0-1
	RBC count	4.52	4.5-5.5million/cumm
	Platelet count	1.40	1.5-4.5lakh/cumm
	ESR (Manual/Automated)	19mm	
	MPV	10.8	7-11fL
	PCV	34	35-46%
	MCV	75.2	80-100fL
	MCH	24.1	27-34pg
	MCHC	32.1	31-36%
	RDW-CV	16.5%	11.5-14.5%
PDW-CV	16.5	10-18%	
BIOCHEMISTRY	Random Blood Sugar	96 mg/dl	70-140 mg/dl
SERUM ELECTROLYTES	Sodium	144 mEq/L	136-145 mEq/L
	Potassium	4.1 mEq/L	3.48-5 mEq/L
	Chloride	103 mEq/L	mEq/L
LIVER FUNCTION TEST	Total Protein	5.1 g/dl	6-8.3 g/dl
	Albumin	3.0 g/dl	3.2-5.4 g/dl
	Globulin	2.1 g/dl	2.5-3 g/dl
	A/G Ratio	1.4	1.2-1.5
	Bilirubin Total	0.4 mg/dl	0.2-1.2 mg/dl
	Bilirubin Conjugated	0.1 mg/dl	0.1-0.4 mg/dl
	Bilirubin Unconjugated	0.3 mg/dl	0.2-0.7 mg/dl

	Alanine Transaminase (ALT/SGPT)	23 U/L	0-45 U/L
	Aspartate Transaminase (AST/SGOT)	58 U/L	0-40 U/L
	Alkaline Phosphatase (ALP)	02 U/L	20-140 U/L
RENAL PROFILE	Blood Urea	17 mg/dl	18-45 mg/dl
	Serum Creatinine	0.8 mg/dl	0.7-1.4 mg/dl
URINE ROUTINE	Urine Albumin	NIL	
	Urine Sugar	NIL	
	Urine Microscopy	NAD	



Figure:01 Showing multiple lichenoid eruptions

DIAGNOSIS:

Ethambutol-Induced Lichenoid Drug eruption in a patient undergoing treatment for pulmonary tuberculosis.

MANAGEMENT:

Immediate cessation of ethambutol from the ATT regimen.

Initiation of an alternative, WHO-recommended anti-tubercular regimen ensuring adequate coverage while preventing treatment interruption.

Oral corticosteroids were administered to attenuate cutaneous inflammation and immune reactivity.

High-potency topical corticosteroids and non-sedating antihistamines were prescribed to mitigate pruritis and facilitate cutaneous healing.

OUTCOME:

Over the course of three weeks, the patient exhibited marked clinical improvement, with regression of active lesions and subsidence of pruritis, leaving behind post-inflammatory hyperpigmentation. She continued therapy with the

modified regimen without recurrence of dermatologic manifestations.

III. DISCUSSION:

TB treatment is associated with a broad range of severe cutaneous ADRs, including LDRs, drug hypersensitivity syndrome, and Stevens-Johnson syndrome. ATT is known to cause a spectrum of cutaneous ADRs. The diagnosis of Ethambutol drug induced lichenoid eruption was based on patient past history, morphology of skin lesions and relationship of cessation of regimen. The underlying mechanism involves T-cell expansion in response to the drug, triggering a delayed-type IV hypersensitivity reaction. Key mediators implicated in this reaction include INF- α , IFN- γ , and CXCR3 ligands.[4] Various drugs cause lichenoid drug eruptions that includes thiazides, antimalarials, diuretics, beta blockers, ACE inhibitors, gold salts, penicillamine and also lipid-lowering drugs i.e. statins and fibrates.

Cutaneous adverse reactions occur in weeks to over a year after the first administration of offender drug. In our case, reaction occurs after one

week of initiation of therapy. Resolution of lesions after the withdrawal of offending drug and remission after reintroduction is a susceptible test for confirming the culprit drug, but it does not always reproduce the skin reaction, though the process is time-consuming for when multiple drugs are suspect. Lichenoid drug eruptions are self-limiting after withdrawal of offending drug. In some patient lesions reoccur even with stopping the offending drug, in such cases management is required.[1] Once drug-induced lichenoid eruptions are detected, treatment involves withdrawing the suspected drug and administering high-dose topical corticosteroids such as fluocinonide or clobetasol. In cases where topical corticosteroids are ineffective, calcineurin inhibitors may be used. Severe and widespread eruptions may require oral prednisone therapy for three to six weeks. Discontinuation of antitubercular therapy poses a considerable threat in the form of disseminated and multi-drug-resistant tuberculosis. In order to mitigate the risk, initiation of an alternative WHO-recommended anti-tubercular regimen is needed.[5] In our study, no new lesions appeared after rechallenge with isoniazid and rifampicin and levofloxacin. However, ethambutol was not reintroduced due to strong suspicion, by exclusion, that it was the offending agent, whereas on rechallenge with isoniazid and rifampicin and levofloxacin, the patient's skin lesions gradually improved with eventual resolution of hyperpigmentation. Strict vigilance after treatment is mandatory as complications such as exfoliative dermatitis, relapse and resistance to tuberculosis can occur. Recording and reporting of cutaneous adverse drug reactions has been weak in the chain of measures to ensure future drug safety. This neglect may be serious later on as re-exposure can have detrimental effects. Furthermore, pharmacovigilance reporting and assessment is needed to harbor the knowledge of the unwanted effects and to raise awareness of medications.[6] Moreover, it underscores the vital role of pharmacovigilance in capturing and analysing such uncommon reactions, ultimately contributing to safer prescribing practices and improved patient care. Encouraging active ADR reporting and strengthening surveillance mechanisms can play a transformative role in reducing preventable drug-related morbidity.

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