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DRUG ERUPTION DUE TO ANTITUBERCULOSIS DRUGS: A CASE REPORT

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Abstract

Drug eruptions due to anti-tuberculosis drugs (ATDs) can compromise treatment adherence and outcomes. Identifying the culprit drug within combination regimens is particularly challenging, especially in patients with comorbidities that may increase susceptibility to cutaneous adverse reactions. We report a case of pulmonary tuberculosis with diabetes mellitus and hypertension who developed a generalized drug eruption after two months of ATD therapy. Clinical data, laboratory findings, management, and follow-up were evaluated descriptively. A 67-year-old male developed a generalized pruritic maculopapular eruption with edema after two months of fixed-dose ATD therapy. Laboratory tests showed no systemic involvement. The regimen was modified by continuing isoniazid and ethambutol while discontinuing rifampicin and pyrazinamide. Supportive therapy with a short course of systemic corticosteroids, antihistamines, and topical agents led to marked improvement within one week. The cautious use of systemic corticosteroids in a tuberculosis patient with comorbidities facilitated rapid resolution without clinical deterioration. This case highlights the need for individualized management of ATD-induced drug eruptions in patients with comorbidities. Selective continuation of isoniazid and ethambutol, withdrawal of rifampicin and pyrazinamide, and careful corticosteroid use proved both effective and safe. This report adds to the limited clinical evidence on managing drug eruptions in high-risk TB patients.

Keywords: **:** *Cutaneous adverse drug reaction, Anti-tuberculosis drugs, Maculopapular rash, Isoniazid, Fixed-dose combination therapy*

Abstrak

Erupsi obat akibat obat anti-tuberkulosis (OAT) merupakan efek samping yang dapat mengganggu keberhasilan terapi. Tantangan terbesar adalah menentukan obat penyebab di tengah terapi kombinasi, khususnya pada pasien dengan komorbid yang dapat memperberat risiko reaksi kulit. Dilaporkan laporan kasus seorang pasien TB paru dengan komorbid diabetes mellitus dan hipertensi yang mengalami erupsi kulit setelah dua bulan terapi OAT. Data klinis, pemeriksaan penunjang, serta tindak lanjut terapi dievaluasi secara deskriptif. Seorang pria 67 tahun mengalami erupsi makulopapular generalisata dengan pruritus dan edema setelah dua bulan terapi OAT kombinasi. Pemeriksaan laboratorium tidak menunjukkan keterlibatan sistemik. Regimen dimodifikasi dengan melanjutkan isoniazid dan etambutol serta menghentikan rifampisin dan pirazinamid. Terapi suportif berupa kortikosteroid sistemik jangka pendek, antihistamin, dan agen topikal menghasilkan perbaikan signifikan dalam satu minggu. Keputusan penggunaan kortikosteroid pada pasien TB dengan komorbid berhasil mempercepat resolusi gejala tanpa memperburuk kondisi dasar. Kasus ini menyoroti pentingnya pendekatan individual dalam menatalaksana erupsi obat akibat OAT, khususnya pada pasien dengan komorbiditas. Strategi selektif melanjutkan isoniazid dan etambutol sambil menghentikan rifampisin dan pirazinamid, ditambah penggunaan kortikosteroid secara hati-hati, terbukti efektif dan aman. Laporan ini menambah bukti klinis mengenai tata laksana erupsi obat pada pasien TB dengan risiko tinggi.

Kata Kunci: Reaksi obat pada kulit, Obat anti tuberkulosis, Ruam makulopapular, Isoniazid, Terapi kombinasi dosis tetap

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Introduction

Globally, tuberculosis (TB) remains a significant public health issue. The World Health Organization (WHO) Global Tuberculosis Report 2024 estimated about 10.8 million new TB cases in 2023, with more than 1.2 million deaths, making TB one of the leading causes of infectious mortality worldwide (Chen et al., 2025). Indonesia is ranked third among high-burden countries after India and China, contributing approximately 10% of global TB cases (Lv et al., 2024). Consequently, the widespread use of anti-tuberculosis drugs (ATDs) is unavoidable, which in turn highlights the clinical importance of drug-related adverse effects.

First-line ATDs include isoniazid, rifampicin, ethambutol, and pyrazinamide, commonly administered in fixed-dose combinations. These regimens are highly effective against *Mycobacterium tuberculosis* but are well documented to cause cutaneous adverse drug reactions (cADRs) (Khandpur & Ahuja, 2022). A prospective study in India involving 3.164 TB patients reported that 1.77% developed cADRs, with maculopapular rash being the most frequent manifestation and ethambutol identified as the most common offending agent upon drug rechallenge (Goel et al., 2025). Similarly, a multicenter cohort study in Morocco including 2.532 TB patients found that 3.7% experienced cutaneous ADRs during treatment (El Hamdouni et al., 2020).

Drug eruptions are among the most frequently observed adverse drug reactions (ADRs) in clinical practice, with a clinical spectrum ranging from mild maculopapular rashes to severe, potentially fatal conditions such as as SJS/TEN. (Owen & Jones, 2021) ; (Cantet et al., 2021). Most of these reactions are idiosyncratic, dose-independent, and typically immune-mediated, particularly involving type IV (delayed) hypersensitivity. Activation of T lymphocytes and the subsequent release of proinflammatory cytokines play a major role in epidermal and mucosal damage (Gupta et al., 2024) ; (Cantet et al., 2021). Recent studies further suggest that maculopapular rash is the most common manifestation of ATD-induced eruptions (Singh et al., 2025).

Despite their frequency, the diagnosis of ATD-induced drug eruptions remains challenging. ATDs are administered in combination therapy, making it difficult to identify the causative agent, while clinical features such as maculopapular rash may mimic viral exanthema or contact dermatitis (Jadhav et al., 2021). Severe reactions can necessitate interruption or discontinuation of TB therapy, potentially reducing adherence, increasing the risk of treatment failure or relapse, and contributing to the emergence of drug-resistant TB (El Hamdouni et al., 2020).

Therefore, reporting case studies of ATD-induced eruptions is crucial to raise clinical awareness, characterize their patterns of presentation, and support the development of safer management guidelines. Early recognition and timely intervention including regimen modification, drug substitution, or rechallenge strategies with supportive therapy—are essential to prevent progression to life-threatening reactions such as SJS/TEN, while safeguarding the success of TB therapy.

However, current literature still lacks clarity on practical approaches to promptly identify the causative agent within combination ATD regimens and ensure treatment continuity without compromising patient safety. This gap highlights the importance of case-based

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evidence that not only illustrates clinical manifestations but also emphasizes strategies for accurate diagnosis and effective management.

To our knowledge, reports describing ATD-induced maculopapular eruptions in elderly tuberculosis patients with comorbid diabetes mellitus and hypertension but without systemic involvement remain scarce. This case contributes novel insight by highlighting a management approach that enabled continuation of therapy with isoniazid and ethambutol while discontinuing rifampicin and pyrazinamide, thereby maintaining treatment effectiveness and ensuring patient safety.

Methods

This study employed a descriptive design in the form of a case report. Clinical data were collected from a single patient through a series of assessments including general examination**,** systemic examination**,** local examination**,** and supporting investigations such as laboratory tests. Additionally, documentation was made regarding the diagnosis, treatment administered, and follow-up outcomes. No specific research intervention was performed; the report merely describes the chronological course of the disease and its clinical management.

The patient provided informed consent for publication of this case and the study was approved by the relevant health research ethics committee. All efforts were made to ensure patient anonymity.

Results

History

A 67-year-old male, Mr. S, was diagnosed with pulmonary tuberculosis two monthssprior to hospital admission. He had been receiving fixed-dose combination (FDC) therapy consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol. Approximately two months into therapy, he developed a reddish rash that first appeared on the upper extremities and subsequently spread to the abdomen and entire body. The rash was characterized by small maculopapular lesions associated with pruritus, progressing within days into dry, scaly desquamation. He also reported edema in both hands and feet. The patient had no history of drug allergy or previous dermatologic conditions. His comorbidities included type 2 diabetes mellitus and hypertension. He was not taking any other medications during ATD therapy.

General examination

On physical examination, the patient appeared weak but alert (compos mentis). Vital signs were stable: blood pressure 140/80 mmHg, heart rate 85 bpm, body temperature 36°C, respiratory rate 25 breaths/min, and oxygen saturation 97% on room air. Body weight was 65 kg, height 170 cm, with a body mass index of 22,5.

Systemic examination

Systemic examination revealed no significant abnormalities.

Local examination

Multiple maculopapular lesions were distributed over the upper and lower extremities, abdomen, chest, and back. The lesions appeared as erythematous macules and papules of small to moderate size, some confluent. Several areas showed desquamation and post-inflammatory hyperpigmentation, with dry and scaly surfaces.

Mild lichenification was noted in certain areas, likely due to repeated scratching from pruritus. No bullae, vesicles, or target lesions were observed. Signs of necrosis, crusting, or active excoriation were absent.

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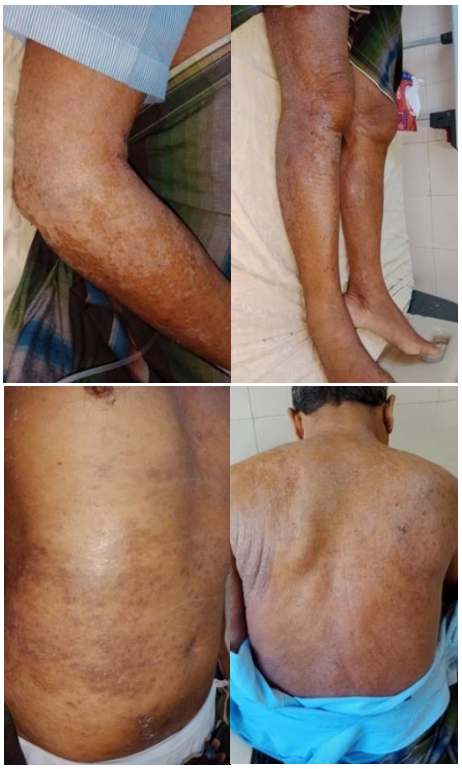

Figure 1. Generalized pruritic maculopapular rash after ATD

Investigations

Hematology revealed mild microcytic normochromic anemia. No leukocytosis, thrombocytosis, or eosinophilia was detected. Neutrophil-to-lymphocyte ratio was within normal limits, with no evidence of systemic inflammation.

Liver and renal function tests were normal, without signs of hepatotoxicity or renal impairment. Electrolyte levels were within physiological ranges. Random blood glucose was 174 mg/dL, without acute hyperglycemia. Overall, laboratory findings indicated stable systemic status without major organ involvement.

Importantly, no other concurrent medications apart from anti-tuberculosis drugs were used, thereby strengthening the causal association between ATDs and the skin eruption.

No specific confirmatory diagnostic procedures, such as skin biopsy, patch testing, or drug rechallenge, were performed; therefore, the attribution of the eruption to anti-tuberculosis drugs was based primarily on clinical presentation and temporal association.

Table 1. Laboratory Investigations

Parameter	Result	Reference Range	Interpretation
Hemoglobin	10.4	13.2-17.3 g/dL	Mild anemia
PCV	30.2	40-52%	Low
RBC	3.940.000	4.2-6.0 x10^6/µL	Low
MCV	76.6	80-100 fL	Microcytic
Other laboratory parameters*	-	-	Within normal limits

*Other laboratory parameters include WBC, Platelets, MCH, MCHC, Absolute neutrophil & lymphocyte counts, NLR, different count, Electrolytes, RFT, LFT, and Blood glucose.

Management

Inpatient Care

ATD regimen was temporarily discontinued and modified based on body weight to isoniazid 300 mg and ethambutol 1000 mg daily. Supportive therapy included intravenous fluids (Asering 1500 cc/24h), intramuscular dexamethasone 5 mg twice daily, intravenous antrain 1 g three times daily, intravenous ranitidine 50 mg twice daily, and intravenous diphenhydramine 10 mg

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twice daily. Topical therapy included desoximetasone 0.25% cream and Vaseline album, applied twice daily.

Outpatient Care

Oral therapy included dexamethasone 0.5 mg once daily and cetirizine 10 mg twice daily. Topical therapy included desoximetasone 0.25% cream and urea 10% cream, applied twice daily.

Patient Education

The patient was advised to maintain skin hygiene with gentle soap, avoid alcohol- or fragrance-containing skincare products, refrain from scratching to prevent irritation or secondary infection, and wear loose clothing to reduce friction on affected skin.

Follow-up

After modification of the suspected ATD regimen and initiation of supportive treatment, the patient showed significant clinical improvement by the first-week evaluation. Erythematous maculopapular lesions diminished, most had resolved, pruritus subsided, and no new lesions developed. The patient's overall condition also improved, with no systemic complaints.

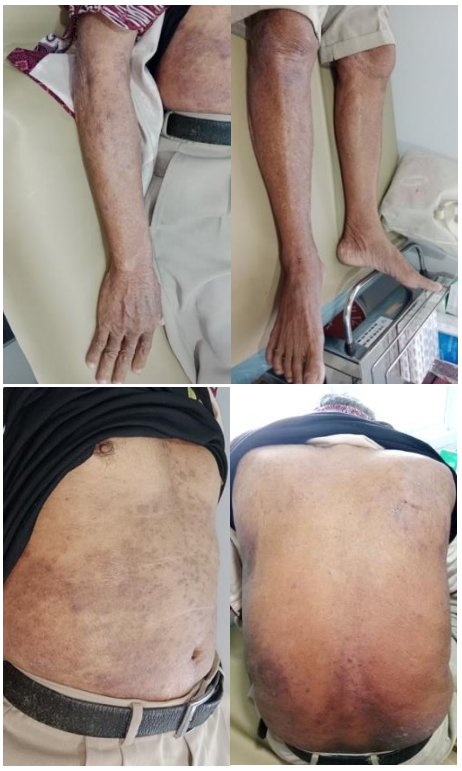

Figure 2. Improvement of rash after treatment

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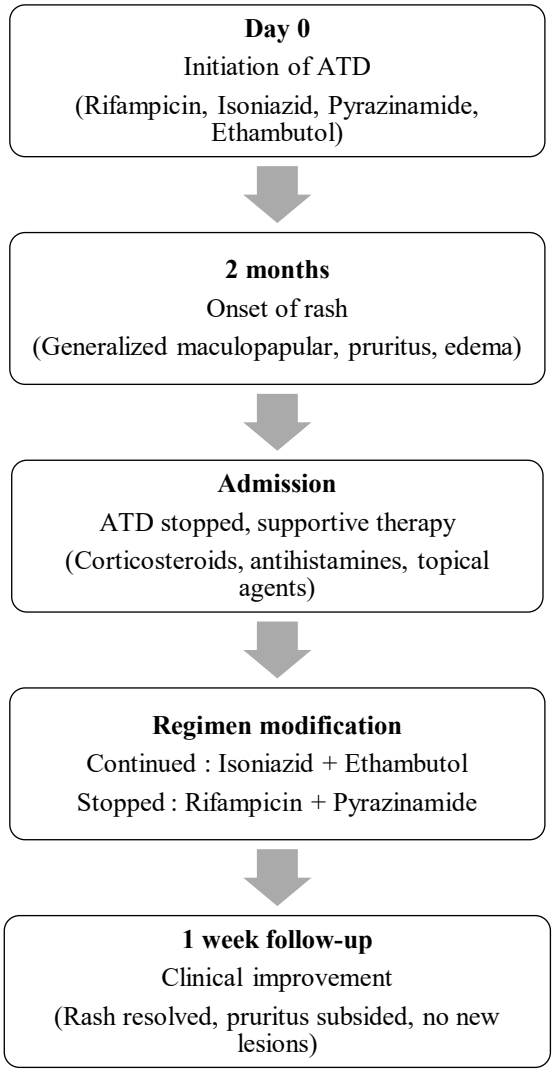

Figure 3. Timeline of clinical events

Discussion

Drug eruptions are hypersensitivity reactions that may occur in response to medications, including anti-tuberculosis drugs (ATDs). These reactions are mediated by immune mechanisms, particularly type IV hypersensitivity involving T-lymphocytes, which trigger inflammatory processes leading to cutaneous manifestations. The variability of immune responses among individuals explains the diversity of drug reactions (Jin et al., 2021).

In this case, the general examination revealed that the patient appeared weak but was fully conscious with stable vital signs. The absence of hemodynamic instability suggests that the drug eruption did not progress to systemic compromise, which is in line with previous studies reporting that most ATD-induced reactions present without acute systemic deterioration (Shepshelovich et al., 2017).

Systemic examination showed no abnormal findings, and laboratory investigations confirmed the absence of leukocytosis, eosinophilia, or hepatic and renal dysfunction. This is important because severe cutaneous adverse reactions such as DRESS or SJS/TEN are frequently accompanied by systemic involvement and hematologic abnormalities. The absence of these findings in our case supports the diagnosis of a mild-to-moderate drug eruption and correlates with prior evidence that most patients improve with supportive therapy, while only a minority require modification or discontinuation of ATD regimens (Miceli et al., 2021).

Local examination demonstrated multiple maculopapular lesions that progressed to dry, scaly desquamation with post-inflammatory hyperpigmentation, but without bullae, vesicles, or mucosal involvement. These clinical features are characteristic of mild-to-moderate drug eruptions and are consistent with previous descriptions of ATD-induced rashes (Muzumdar et al., 2019). Importantly, the absence of target lesions, necrosis, or mucosal damage helped exclude severe conditions such as SJS or TEN. The mild lichenification observed in certain areas was likely secondary to persistent pruritus, a common finding in chronic or resolving drug eruptions.

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Investigative approaches further reinforced the diagnosis. Laboratory results showed only mild microcytic normochromic anemia, with no systemic inflammation, hepatotoxicity, or renal impairment. These findings excluded alternative etiologies such as viral exanthems or systemic drug reactions, in which laboratory abnormalities are more common (Cook & Décary, 2020). Thus, the role of investigative methods was crucial in differentiating drug eruption from other dermatological or systemic conditions.

The temporal relationship between the initiation of ATD therapy and the onset of the rash strongly supports ATD-induced eruption, particularly as the patient developed symptoms after two months of treatment. This aligns with previous studies reporting that most eruptions occur within the first two months of therapy (Shepshelovich et al., 2017). In this case, the clinical decision to continue isoniazid and ethambutol while discontinuing rifampicin and pyrazinamide was based on both clinical judgment and previous evidence, as isoniazid and ethambutol are less frequently associated with severe cutaneous reactions compared to rifampicin and pyrazinamide (Sharma et al., 2020). This strategy enabled treatment continuity while minimizing the risk of recurrent or more severe eruptions.

The use of systemic corticosteroids in drug eruptions remains controversial, particularly in TB patients. Although systemic corticosteroids carry the risk of suppressing host immunity, they may be justified in selected cases to control severe inflammation and prevent further tissue damage (Hama et al., 2022). In our patient, a short course of systemic corticosteroids was administered alongside antihistamines and topical corticosteroids, resulting in significant clinical improvement without systemic deterioration. This outcome highlights the importance of individualized risk–benefit assessment when considering corticosteroid therapy in TB patients with drug eruptions.

Additionally, the presence of comorbid diabetes mellitus and hypertension in this patient deserves consideration. Several recent studies suggest that diabetes may increase susceptibility to adverse cutaneous drug reactions, possibly due to altered immune regulation and chronic systemic inflammation (Shi et al., 2024). Similarly, comorbid conditions such as hypertension can complicate overall drug metabolism and therapeutic decision-making, even if not directly causal for skin eruptions (Chidambaram et al., 2021). The absence of systemic abnormalities in this patient, despite these comorbidities, underscores the relatively mild course of the reaction and highlights the importance of individualized assessment in patients with multiple risk factors.

Patient education focusing on skincare, avoidance of irritants, and adherence to follow-up was also emphasized. Awareness of potential drug reactions and close monitoring reduce recurrence risk and enable early detection of systemic complications (Fahad Alhassoon et al., 2022). Overall, this case underscores the importance of thorough clinical evaluation, including general, systemic, local, and investigative approaches, to establish the correct diagnosis of ATD-induced drug eruptions.

Importantly, this report contributes to the existing literature by illustrating a case of generalized maculopapular eruption in an elderly TB patient with comorbid

diabetes and hypertension but without systemic involvement or laboratory abnormalities. The clear chronological sequence of drug exposure, exclusion of other causes, and improvement after regimen modification strengthen the diagnosis. Moreover, the management strategy—selectively continuing isoniazid and ethambutol while withdrawing rifampicin and pyrazinamide—demonstrates a practical approach to balancing effective TB therapy with patient safety. Such evidence is valuable in guiding clinicians facing similar diagnostic and therapeutic challenges.

Conclusions

Cutaneous drug eruptions from anti-tuberculosis therapy represent a significant clinical challenge, particularly in elderly patients with comorbidities. This case illustrates a generalized maculopapular eruption developing two months into therapy, which was effectively managed through selective continuation of isoniazid and ethambutol, withdrawal of rifampicin and pyrazinamide, and supportive treatment including short-course corticosteroids. The absence of systemic involvement and the rapid resolution of symptoms emphasize the importance of timely recognition and tailored regimen modificasion to maintain treatment efficacy while ensuring patient safety. This case further underscores the need for heightened vigilance, especially in patients with diabetes and hypertension, and contributes practical insight into balancing effective TB control with the management of drug-induced adverse reactions.

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