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(CASE REPORT)

Antituberculosis drug-induced toxicoderma: A rare but potentially severe adverse reaction: A case report

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Abstract

We report the case of a 52-year-old male patient with pulmonary tuberculosis who, 72 hours after initiating antituberculosis therapy, developed a severe drug-induced toxicoderma. The clinical presentation was characterized by intense pruritus, erythematous rash, eczema, and diffuse skin desquamation with a positive Nikolsky sign. Given the suspicion of a severe adverse reaction, the antituberculosis regimen was immediately discontinued, and management with fluid support and topical corticosteroids was initiated. Skin biopsy revealed a perivascular lymphoplasmacytic infiltrate, lymphocytic exocytosis, and neutrophilic aggregates, findings consistent with a reactive inflammatory process induced by drugs.

The discussion focuses on the immunological complexity of the case, which exhibited overlapping clinical and histological features between type IVc (cytotoxic) and type IVd (neutrophilic) hypersensitivity reactions, suggesting a combined form within the spectrum of toxic epidermal necrolysis (TEN) and acute generalized exanthematous pustulosis (AGEP). In addition to cutaneous involvement, the patient developed a mixed pattern of liver injury that resolved following drug withdrawal. After complete remission of the lesions was achieved, a stepwise and monitored reintroduction of the regimen was carried out, which was well tolerated.

This case underscores that early diagnosis and prompt discontinuation of the causative agent are the most critical prognostic factors in preventing fatal outcomes in drug-induced toxicodermas associated with first-line antituberculosis medications.

Keywords: Toxicoderma; Pulmonary tuberculosis; Antituberculosis drugs; Nikolsky sign; Delayed hypersensitivity; Skin biopsy; Drug reintroduction

1. Introduction

Toxicodermas are defined as adverse mucocutaneous manifestations caused by the administration of substances, generally medications, via any route (topical, oral, or parenteral) [1,2]. These reactions are frequent reasons for consultation in clinical practice, spanning a wide spectrum from mild exanthemas to life-threatening forms such as Stevens-Johnson Syndrome (SJS) or Toxic Epidermal Necrolysis (TEN) [2,3].

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In the context of tuberculosis treatment, first-line drugs (isoniazid, rifampin, pyrazinamide, and ethambutol) are recognized agents for triggering delayed hypersensitivity reactions [4]. Diagnosis is primarily clinical, based on lesion morphology and the chronology between drug administration and symptom onset [5,6]. However, identifying the causative agent remains a challenge, particularly in patients receiving polypharmacy. This requires a high index of suspicion and close monitoring to avoid recurrences or severe visceral complications, such as DRESS syndrome [3,6].

Early diagnosis and immediate withdrawal of the culprit drug are fundamental pillars for reducing morbidity, mortality, and long-term sequelae [5]. Given the necessity of maintaining antituberculosis treatment for infection control, managing these dermatological complications requires a rigorous approach that balances patient safety with therapeutic efficacy. The objective of this article is to report a case of antituberculosis drug-induced toxicoderma, analyzing its semiological presentation and the diagnostic-therapeutic approach adopted.

2. Case Presentation

A 52-year-old male with a confirmed diagnosis of pulmonary tuberculosis was undergoing standard antituberculosis treatment. The patient was admitted to the emergency department with an acute clinical picture that began approximately 72 hours after the first dose of medication. Symptoms started with intense generalized pruritus, rapidly progressing to an erythematous exanthema with eczema and diffuse cutaneous desquamation involving extensive body areas. Physical examination revealed predominant desquamative lesions on the trunk and lower extremities (Images 1 and 2), notably featuring a positive Nikolsky sign. This finding suggested severe compromise of dermo-epidermal cohesion, raising high clinical suspicion of severe drug-induced toxicoderma.

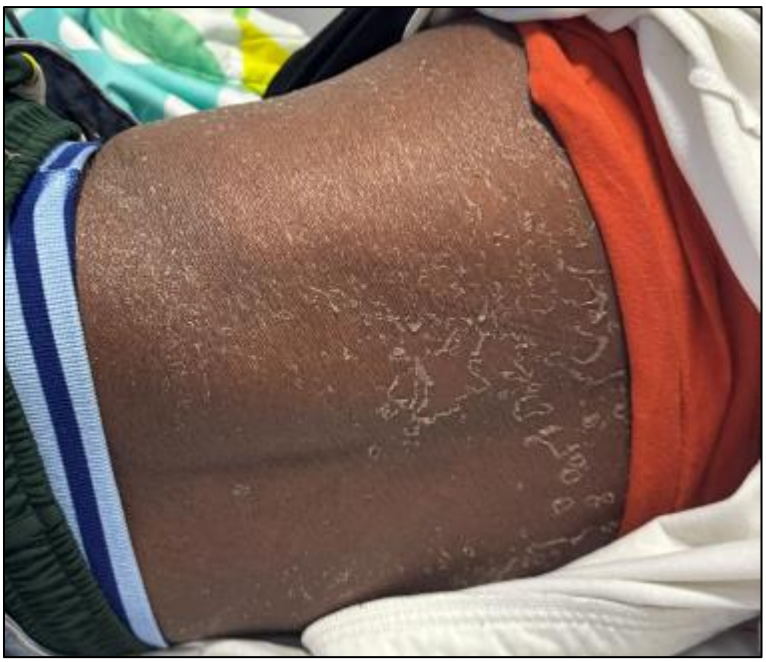

Figure 1 Diffuse desquamation on trunk

Figure 2 Desquamation on lower limbs

Given the severity and imminent risk of progression, antituberculosis therapy was immediately suspended. Comprehensive supportive care was established, including intravenous fluid therapy for hemodynamic stability and medium-potency topical corticosteroids. A skin biopsy was performed for diagnostic confirmation. Histopathological study confirmed a stratum corneum with serous exudate and neutrophil clusters, alongside a stratum spinosum showing reactive changes and lymphocyte exocytosis. The dermis exhibited marked edema associated with a moderate chronic, perivascular, lymphoplasmacytic inflammatory infiltrate; additionally, permeation of blood vessels by histiocytes and neutrophils was noted in the absence of vascular fibrinoid necrosis.

These morphological findings confirmed a reactive inflammatory process compatible with pharmacoderma. During hospitalization, the patient developed a mixed pattern of hepatic injury, which resolved favorably with supportive management. Cutaneous evolution was successful, with gradual remission of pruritus and re-epithelialization of desquamative areas. Once stabilized, a sequential, graded drug reintroduction was performed under strict hospital monitoring. The protocol was tolerated satisfactorily without recurrence of dermatological manifestations, allowing for the continuation of the primary pulmonary treatment with a favorable short-term prognosis.

3. Discussion

Regarding cutaneous adverse drug reactions (CADR), Trujillo and Vásquez [7] note that these are frequent hospital complications, with antimicrobials—including antituberculosis agents—being predominant etiological factors. In this case, the onset of symptoms 72 hours post-initiation suggests a T-cell-mediated delayed hypersensitivity mechanism. According to the Lerch and Pichler subclassification [8], this case fits within Type IV reactions, specifically Type IVc, where cytotoxic T-lymphocytes and effector proteins (perforin/granzyme B) orchestrate keratinocyte apoptosis, explaining the desquamation and positive Nikolsky sign.

However, the histological presence of neutrophil clusters and serous exudate introduces diagnostic complexity, as these are characteristic of Type IVd reactions (such as AGEP), where T-cell IL-8 secretion induces granulocyte recruitment [8]. This clinical and histopathological duality supports the hypothesis of "combined" or overlapping forms discussed by Horcajada-Reales et al. [9]. These authors argue that severe toxicodermas do not always present as pure entities but may exhibit overlapping features between the SJS/TEN spectrum and reactions like AGEP.

The severity of the tegumentary involvement necessitated the immediate suspension of the antituberculosis regimen, which remains the most decisive prognostic factor in managing severe toxicodermas [7,9]. The successful graded reintroduction demonstrates the importance of a multidisciplinary approach to identify immunological tolerance to the suspected drug's metabolites.

4. Conclusion

From a clinical perspective, this report reaffirms that early identification and immediate interruption of suspected drugs are the most critical prognostic factors for survival and the limitation of sequelae. In high-impact public health pathologies like tuberculosis, where treatment continuity is imperative, drug suspension must be complemented by rigorous supportive care and a strictly monitored reintroduction strategy. This procedure distinguishes between permanent hypersensitivity and transient reactions, ensuring patient safety without compromising long-term therapeutic success**.**

Compliance with ethical standards

The authors declare that there are no conflicts of interest regarding the publication of this article. They report no financial, personal, or professional relationships that could have influenced the study design, data collection, analysis, interpretation, or the writing of this manuscript. Written informed consent was obtained from participants included in this study prior to their involvement. The consent process ensured that each participant was fully informed about the nature and purpose of the study, as well as the use of their clinical information for research and publication, in accordance with ethical standards.

References

[1] Alvargonzález M, Gijón Conde T. Toxicoderma: a case report. Semergen. 2019;45(3):e13–e14.

[2] Bourrain JL. Toxicodermas and drug-induced cutaneous hypersensitivity reactions. EMC - Dermatology. 2025;27(1):1–14.

[3] Duong T, Valeyrie-Allanore L. Toxicodermas. EMC - Medicine. 2010;14(1):1–13.

[4] Luyasu S, Beaudouin E, Kanny G. Drug hypersensitivity reactions. EMC - Medicine. 2021;25(2):1–5.

[5] Gómez de la Fuente E, Pinedo Moraleda F, Griffiths Acha J. Toxicodermas: from clinical features to histology. Piel (Barc). 2025;40(7):449–455.

[6] Skayem C, Duong TA. Toxicodermas. EMC - Medicine. 2025;29(3):1–6.

[7] Trujillo MC, Vásquez LA. Clinical and epidemiological characteristics of toxicodermas in hospitalized patients at Hospital Pablo Tobón Uribe, Medellín, Colombia, 2007–2009. Revista de la Asociación Colombiana de Dermatología. 2012;20(3):281–296.

[8] Lerch M, Pichler WJ. The immunological and clinical spectrum of delayed drug-induced exanthems. Current Opinion in Allergy and Clinical Immunology. 2004;4(5):411–419.

[9] Horcajada-Reales C, Pulido-Pérez A, Suárez-Fernández R. Severe toxicodermas: do combined forms exist? Actas Dermo-Sifiliográficas. 2016;107(1):23–33.