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Case Report

When Treatment Turns Fatal: Lyell's Syndrome Induced by Sulfasalazine - A Case Report

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Abstract

A 67-year-old woman with ulcerative colitis developed a generalized mucocutaneous eruption six weeks after starting sulfasalazine. Examination revealed widespread epidermal detachment involving approximately 40% of the body surface area, with extensive erosions affecting the limbs, back, buttocks, and hands, associated with oral, ocular, vulvar, and perianal involvement. Laboratory findings included anemia, leukocytosis, elevated C-reactive protein, hyperkalemia, renal impairment, markedly elevated D-dimer levels, and rhabdomyolysis. Her SCORTEN was 4, corresponding to an estimated mortality of 58.3%. Histopathological examination demonstrated keratinocyte necrosis, subepidermal detachment, and a dermal perivascular lymphocytic infiltrate, confirming toxic epidermal necrolysis. Sulfasalazine was immediately discontinued, and intensive supportive care was initiated. Despite treatment, the patient developed fever, altered consciousness, and progressive skin lesions. She was transferred to the intensive care unit and subsequently died from multiorgan failure. This case highlights the potentially fatal risk of sulfasalazine-induced toxic epidermal necrolysis.

Introduction

Toxic epidermal necrolysis (TEN) is a rare, potentially life-threatening drug-induced condition [1,2]. Sulfasalazine, a disease-modifying agent widely prescribed for ulcerative colitis and rheumatoid arthritis for more than four decades, is an uncommon but recognized cause of severe cutaneous adverse reactions, including Stevens–Johnson syndrome (SJS) and TEN. Despite its widespread use, only a limited number of sulfasalazine-induced TEN cases have been described in the literature [1]. We report a case of sulfasalazine-induced TEN.

Case Report

A 67-year-old woman with a history of ulcerative colitis, treated with sulfasalazine for six weeks, presented with a generalized mucocutaneous eruption evolving for five days prior to admission.

Physical examination revealed extensive epidermal detachment involving approximately 40% of the body surface area, characterized by widespread erosive plaques on an erythematous background, predominantly affecting the limbs (Figure 1A), back, buttocks (Figure 1B), and dorsal surfaces of the hands (Figure 1C), in association with vulvar (Figure 2A), perianal (Figure 2B), oral mucosal (Figure 2C) and ocular involvement (Figure 2D).

Laboratory investigations showed normocytic normochromic anemia, leukocytosis, elevated C-reactive protein (54 mg/L), hyperkalemia (5.8 mmol/L), normal natremia, impaired renal function, markedly elevated D-dimer levels (8680 ng/mL), negative troponin, and rhabdomyolysis at five times the normal level.

The patient's SCORTEN was 4 (age >40 years, epidermal detachment >10%

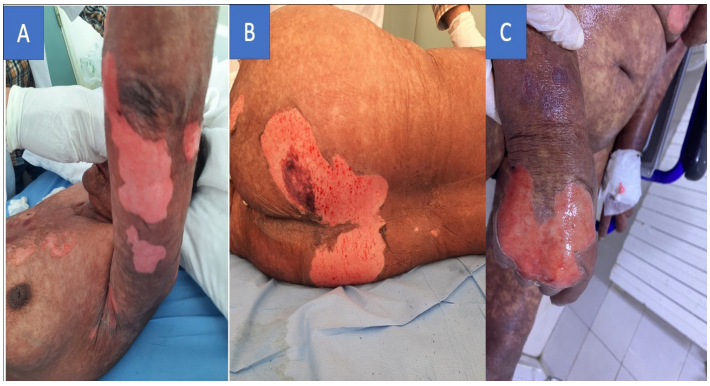

Figure 1: *Widespread epidermal detachment with a characteristic wet-cloth appearance, forming multiple erosive plaques on an erythematous background involving the limbs (A), the back and buttocks (B), and the dorsal surfaces of the hands (C).*

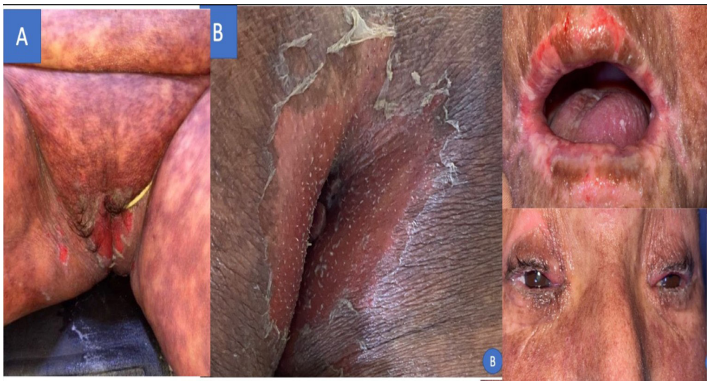

Figure 2: *Sharply demarcated erythematous erosive plaques with irregular borders involving the vulvar (A) and perianal regions. Oral mucosal involvement showing erosive cheilitis affecting both the upper and lower lips (C), and ocular involvement with conjunctival hyperemia and suprapalpebral epidermal detachment (D).*

of body surface area, serum urea >10 mmol/L, and serum bicarbonate <20 mmol/L), corresponding to an estimated mortality of 58.3%.

Sulfasalazine was discontinued. The patient received optimal supportive care, including intensive clinical monitoring, fluid and electrolyte management, meticulous skin and mucosal care, and topical wound treatment.

Skin biopsy demonstrated epidermal keratinocyte necrosis with subepidermal detachment and a perivascular lymphocytic inflammatory infiltrate in the dermis.

The diagnosis of toxic epidermal necrolysis was established based on the compatible six-week interval between sulfasalazine initiation and symptom onset, together with the clinical, laboratory, and histopathological findings.

The clinical course was marked by deterioration of consciousness, a fever spike of 39.5°C, and progression of erosive lesions. The patient was transferred to the intensive care unit, where she subsequently died from multiorgan failure.

Discussion

Toxic epidermal necrolysis is a rare and severe form of drug-induced skin reaction, characterized by widespread epidermal detachment and severe involvement of one or more mucosal surfaces, secondary to massive keratinocyte apoptosis [1].

Common clinical features include blisters and/or erosions preceded by nonspecific symptoms such as erythema, conjunctivitis, enanthema, and fever. Patients may experience mucosal burning, cutaneous pain, cough, palmoplantar erythema, atypical target-like lesions, and purpura, with initial epidermal detachment involving the face and trunk within the first 48 hours. In the established phase, epidermal detachment appears as “wet linen” with a positive Nikolsky sign [3]. The onset typically occurs 4 to 28 days after initiation of the suspected drug [3]. In our case, the delay was one month and five days.

Disease severity is primarily assessed using the SCORTEN, the most widely validated prognostic score for SJS/TEN. Calculated within the first 24 hours of admission and repeated on day 3, it incorporates seven independent risk factors and accurately predicts in-hospital mortality, thereby guiding patient triage and the intensity of supportive care. Our patient had a SCORTEN of 4, corresponding to a predicted mortality of 58.3%, consistent with the unfavorable clinical outcome [4].

The pathophysiology of toxic epidermal necrolysis is complex and not fully understood. Current evidence suggests a drug-specific, immune-mediated cytotoxic reaction involving CD8+ T lymphocytes and natural killer cells. These cells release mediators such as granulysin, perforin, and granzyme B, leading to widespread keratinocyte apoptosis. Additional mechanisms, including Fas–Fas ligand interaction and tumor necrosis factor-alpha, may also contribute [5].

Common causative drugs include sulfonamides, beta-lactams, tetracyclines, quinolones, anticonvulsants, antiretrovirals, nonsteroidal anti-inflammatory drugs, and allopurinol [1].

Despite being a well-established trigger of severe cutaneous adverse reactions, sulfasalazine-induced toxic epidermal necrolysis is exceptionally uncommon, and only a few cases have been documented in the literature [1,2,5].

Management relies on immediate withdrawal of the offending drug and supportive care, including high-protein nutrition, thermal regulation, and aseptic local wound care.

The use of specific therapies remains controversial. Systemic corticosteroids may reduce inflammation and keratinocyte apoptosis, but their benefit is debated due to the risk of infectious complications. Although

several therapies, such as intravenous immunoglobulins, cyclosporine, and anti-TNF agents, have been proposed, no consensus has been established regarding their efficacy, and supportive care remains the cornerstone of treatment [6].

The prognosis remains poor, with a mortality rate of approximately 30% and a significant risk of sequelae [3].

Conclusion

Sulfasalazine-induced toxic epidermal necrolysis is a rare but potentially fatal adverse drug reaction. Our case illustrates the fulminant course and poor prognosis of this condition, culminating in death despite appropriate supportive management. Awareness of this uncommon complication is crucial, as early diagnosis and immediate drug withdrawal remain the most important measures to limit disease progression and reduce mortality.

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