Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Gastroenterology

Dress Syndrome in a Patient Being Treated for Ulcerative Colitis: A Rare Case Induced by Mesalazine

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i06.041 | **Received:** 06.05.2026 | **Accepted:** 13.06.2026 | **Published:** 19.06.2026

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Abstract **Case Report**

DRESS syndrome (Drug Reaction with Eosinophilia and Systemic Symptoms syndrome) is a rare but serious drug reaction with the potential for severe multiorgan involvement, particularly hepatic. It is a drug-induced syndrome that can be life-threatening due to the onset of severe visceral manifestations. It is characterized by a long latency period between drug administration and the onset of symptoms, and by a prolonged course often marked by flare-ups even after discontinuation of the causative drug. Its pathophysiology remains incompletely understood and involves reactivation of herpes viruses (HHV-6, HHV-7, EBV, CMV) and a strong immune response directed against these viruses. The drugs may act both on epigenetic control to promote viral reactivation and on an antiviral T-lymphocyte (T-lymphocyte) immune response by interacting with the major histocompatibility complex receptor in genetically predisposed individuals.

Keywords: Dress syndrome, ulcerative colitis, 5-ASA, mesalazine, skin rash, major cytolysis.

INTRODUCTION

Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD) limited to the mucosa, continuously affecting the rectum and colon. It progresses in flare-ups interspersed with remissions. Its origin is not yet fully understood, but is likely multifactorial, resulting from an immune system dysregulation in response to environmental factors and the gut microbiota, occurring in genetically predisposed patients [1]. Its diagnosis is based on a combination of clinical, laboratory, endoscopic, and histological findings. The choice of treatment strategy and its monitoring depend on the location and severity of the disease. The gastroenterologist plays a central role in patient support and education, follow-up, adherence, detection of drug interactions, and monitoring of adverse effects in these patients.

PATIENT AND OBSERVATION

This is a 40-year-old patient with no significant medical history who is being followed for left-sided ulcerative colitis, presenting with bloody, mucoid diarrhea associated with rectal syndrome. Colonoscopy revealed an erythematous and granular appearance of the mucosa with superficial ulcerations, extending from the anorectal junction to the left colic angle without any interval of healthy mucosa.

Pathological examination of the colonic biopsies revealed distortion of the glandular architecture, cryptitis, cryptic abscesses, and basal plasmacytosis.

The patient was initially prescribed 5 ASA (mesalazine), which he took for one month before discontinuing the medication on his own following the onset of a generalized febrile rash and the persistence of bloody mucoid diarrhea.

Three weeks later, the patient was admitted to our Hepatogastroenterology Department at Arrazi Hospital, part of the Mohammed VI University Hospital in Marrakech, for management of his UC flare-up and cutaneous manifestations.

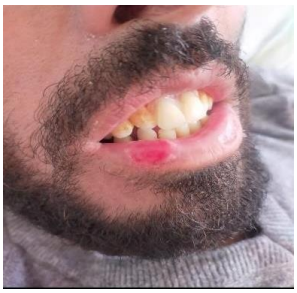
The clinical examination revealed a low-grade fever of 38.2°C and a maculopapular rash primarily affecting the face, trunk, and upper limbs, with the appearance of a vesicular lesion on the lower lip. The remainder of the examination was unremarkable.

Laboratory tests revealed: leukocytosis (22 × 10^5/L), eosinophilia (1,700/mm³), and liver enzyme abnormalities (ALT six times the upper limit of normal, AST five times the upper limit of normal). A systematic evaluation for involvement of other organs was performed, particularly renal, pulmonary, cardiac, and neurological involvement. The remainder of the laboratory workup was unremarkable. Serology for hepatitis A, B, and C, CMV, EBV, and HIV was all negative.

The diagnosis of Dress Syndrome was made based on a body of evidence using the Japanese criteria (Table 1), and treatment consisted of initiating intravenous corticosteroid therapy (1 mg/kg/day) and antiviral therapy with Ganciclovir (mg/kg), along with close clinical and laboratory monitoring. The course of the disease was favorable, marked by rapid normalization of liver function tests (Day 5), a gradual decrease in white blood cells and eosinophils that normalized by Day 10 of treatment, and the complete resolution of skin lesions. IV corticosteroid therapy was switched to oral administration, and the patient was discharged.

Mesalazine was permanently discontinued, and the patient was subsequently placed on azathioprine as maintenance therapy for his UC.

No recurrence has been observed since this episode, with a follow-up of 6 months.



DISCUSSION

DRESS syndrome is a severe drug reaction that occurs 3 to 6 weeks after the administration of the triggering medication.

The diagnosis of DRESS syndrome was made according to the criteria of the Japanese study group (Table 1).

Tableau 1. Critères diagnostiques du DRESS syndrome (Groupe d'études japonais)

○ **Exanthème maculopapuleux > 3 semaines après prise d'un médicament à risque**
○ **Manifestations cliniques persistant > 2 semaines après l'arrêt du médicament**
○ **Fièvre (> 38°C)**
○ **Polyadénopathies**
○ **Elévation des transaminases (ALAT > 100 U/l)**
○ **Une des anomalies suivantes du nombre des leucocytes :**
⊣ Hyperleucocytose > 11G/l
⊣ Lymphocytose atypique (> 5 %)
⊣ Hyperéosinophilie > 1,5G/l
○ **Réactivation HHV-6**
○ **5 critères : DIHS atypique**
○ **7 critères : DIHS typique**

The patient met five of the seven criteria established by the study group. Indeed, viral reactivation (HHV-6, CMV, HHV-7, HSV) has been strongly implicated in the pathophysiology of DRESS syndrome, as such reactivations have been observed in several patients monitored for DRESS syndrome, particularly in cases of HHV-6 infection [6]. Since 2007, viral reactivation of HHV6 has been adopted as a diagnostic

criterion for DRESS syndrome in Japan [3], whether it involves primary infection or viral reactivation, as is the case with our patient. Viral infection appears to act as a cofactor in the course of DRESS syndrome. However, the sequence of events remains controversial: Is viral reactivation the consequence of drug allergy, or is viral reactivation responsible for all the clinical signs of DRESS syndrome? The detection of viral reactivation (as in our patient) justifies the initiation of antiviral treatment with Ganciclovir or Cidofovir.

The role of sulfasalazine in DRESS syndrome is indisputable, given several similar published observations [4]. The main drugs responsible for DRESS syndrome are: phenobarbital, carbamazepine, sodium valproate, and sulfasalazine. Sulfasalazine is composed of a 5-ASA molecule and a sulfapyridine molecule. Due to the sulfapyridine component, it may have a systemic antiinflammatory effect, hence its use in joint diseases.
In contrast, DRESS syndrome induced by 5-ASA is much less frequently described.

CONCLUSION

DRESS warrants prolonged monitoring due to the risk of progressive flare-ups linked to sequential reactivations of herpes viruses. Particular vigilance is required when introducing new treatments, as is often the case for patients treated for epilepsy who require a change in antiepileptic medication. Cross-reactions are possible between molecules of unrelated biochemical nature, illustrating the distinct mechanism of DRESS compared to other drug rashes and a possible direct effect of certain medications on viral biology. The occurrence of delayed relapses is not uncommon, even in the absence of any new medication, particularly during the tapering of systemic corticosteroid therapy, which should be gradual

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