ISSN: 2755-0117

Journal of Oncology Research Reviews & Reports

SCIENTIFIC Research and Community

Case Report

Open Access

A Relapsing Case of Drug Reaction with Eosinophilia and Systemic Symptoms (Dress) Syndrome

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ABSTRACT

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a severe and potentially fatal adverse hypersensitivity reaction often secondary to therapeutic medications. There is a wide variation in the incidence of the disease and is dependent on the genetic makeup of the individual. The most common presentation includes skin eruptions, fever, generalized lymphadenopathy, eosinophilia, and internal organ involvement (most commonly, liver, kidneys, and lungs). Some less common features are dysphagia, agranulocytosis, and chylous ascites. The most common drugs causing DRESS syndrome include carbamazepine, allopurinol, sulfasalazine, phenobarbital, and lamotrigine. Differential diagnosis has Steven-Johnson Syndrome/Toxic Epidermal Necrolysis, hypereosinophilic syndrome, and Sezary syndrome. Systemic corticosteroids are the first-line treatment for DRESS syndrome and the withdrawal of the offending agent and supportive therapy. We report a rare case of DRESS syndrome following the use of clarithromycin and moxifloxacin, where rapid steroid taper resulted in relapse.

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Received: June 16, 2021; **Accepted:** June 23, 2021; **Published:** June 30, 2021

Introduction

Drug reaction with eosinophilia and systemic symptoms [DRESS] syndrome is an idiosyncratic drug reaction with a prolonged latency period and is sometimes referred to as drug-induced hypersensitivity syndrome. The syndrome was initially termed drug-induced pseudolymphoma by Satlztein due to the lymphomatous appearance of lymph nodes in a patient with generalized lymphadenopathy after using hydantoin. It was later termed anticonvulsant induced hypersensitivity syndrome and was observed secondary to carbamazepine use, where patients presented with fever, generalized rash, and lymphadenopathy. The term DRESS was coined by, based on the retrospective study by. The estimated incidence ranges between 1 in 1000 to 1 in 10,000 drug exposures.

It is a potentially life-threatening syndrome with skin eruptions, fever (100.4 to 104°F), diffuse lymphadenopathy, and visceral organ involvement, which may manifest as hepatitis, acute interstitial nephritis, pancreatitis, eosinophilic myocarditis, etc. The time to onset of symptoms after exposure varies among different drugs and ranges from 3 to 105 days. Immediate withdrawal of the causative agent and administration of systemic corticosteroids are the mainstay of treatment. Adequate supportive therapy with antihistamines, antipyretics, and topical emollients are also given. Immunosuppressants or plasmapheresis may be required in severe cases. We present a rare case of DRESS syndrome, which resulted from the use of clarithromycin and moxifloxacin, which was managed with steroids but had relapses due to the rapid tapering of prednisone [1-7].

Case Presentation

A 37-year-old male with no significant past medical history presented to the ED with chief complaints of cough, chest congestion, and wheezing for the past week but denied any further complaints. He was noted to be saturating at 100% on room air and was noted to have coarse breath sounds in the left lower lung base. Chest X-ray showed left lower lobe pneumonia, and the patient was discharged on clarithromycin but was switched to moxifloxacin after three days due to a lack of clinical improvement. After 24 hours, he presented with fever, chills and reported a new onset rash on his torso that spread to the upper and lower extremities.

He was transitioned back to clarithromycin due to concern for moxifloxacin allergy. However, he was hospitalized after a headache, arthralgias, worsening rash, and shortness of breath. On exam, he was febrile to 101°F and tachycardic to 120/min. A diffuse, morbilliform, non-blanching rash was noted on his extremities and torso. Multiple cervical lymph nodes and inguinal lymph nodes were palpable, and diffuse expiratory wheezing was auscultated. Labs showed a WBC count of 29,000 K/uL (n= 4-11.0) with 50 % eosinophilia, atypical lymphocytes, and an elevated alkaline phosphatase elevation on liver function tests.

The viral panel was remarkable for elevated titers of EBV and HHV-6. CT chest demonstrated diffusely enlarged cervical and axillary lymph nodes and small airway bronchiolitis. He was diagnosed with DRESS syndrome as per RegiSCAR criteria. Clarithromycin was discontinued, and the patient was discharged on 60 mg of prednisone taper over two weeks. However, on decreasing his dose by 20 mg, the patient was readmitted to

the hospital for cough, shortness of breath, wheezing, persistent elevation of liver enzymes. The patient was restarted on a high-dose regimen of prednisone 70 mg daily with a slow taper over six to eight weeks. The symptoms subsided, and the patient remained asymptomatic on follow-up with no further relapses.

DRESS syndrome is a life-threatening hypersensitivity reaction that includes an extensive skin rash, eosinophilia, atypical lymphocytosis, generalized lymphadenopathy, and internal organ involvement (liver, kidney, and lungs are most commonly involved) [3]. Several drugs, particularly anticonvulsants and sulfonamides, are known to cause DRESS syndrome [Table 1]. A systematic review of literature by Cacoub et al. described 44 drugs known to cause DRESS syndrome and listed carbamazepine, allopurinol, sulfasalazine, phenobarbital, lamotrigine, and nevirapine as the most commonly associated drugs [5]. Our report describes an unusual case where exposure to clarithromycin and moxifloxacin led to DRESS syndrome.

Table 1

Organ system	Presenting Symptoms
Skin	Urticaria, maculopapular rash, Vesicles, Bullae, pustules, cheilitis, purpura, target lesions, erythroderma, facial edema
Hematologic	Leukopenia, Leukocytosis (atypical lymphocytes), Eosinophilia,
Hepatic	Fulminant hepatic failure, Transaminitis
Renal	Asymptomatic hematuria, proteinuria, elevated BUN, Creatinine, Acute Interstitial nephritis
Lung	Acute interstitial pneumonitis, pleuritis, Acute respiratory distress syndrome
Cardiac	Myocarditis
Neurological	Meningitis, Encephalitis
Gastrointestinal	Gastirits, Colitis, pancreatitis.

Table 2

Category of Drug	Drug name
Anti-microbials	Ampicillin, dapsone, isoniazid, linezolid, minocycline, rifampin, Vancomycin, Trimethoprim sulfa-methoxazole, Pyrimethamine
Antidepressants	Fluoxetine
Anti-convulsant	Carbamazepine, Phenytoin, Lamotrigine, Phenobarbital
Anti-viral	Abacavir, Nevirapine
Antihypertensives	Captopril
Other	Allopurinol, Sulfapyridine

DRESS is now recognized as one of the Severe Cutaneous Adverse Reactions (SCAR) and may have a delayed onset of symptoms with persistence despite the offending agent's withdrawal [5,9,10]. The European Registry of Severe Cutaneous Adverse Reactions (RegiSCAR) has devised a scoring system based upon the clinical features, the extent of skin involvement, organ involvement, and clinical course, and the patients are classified as definite, probable, or possible cases of DRESS [11]. The differential includes Steven Johnson Syndrome, Toxic Epidermal Necrolysis, acute generalized exanthematous pustulosis, and Sezary syndrome (cutaneous T-cell lymphoma with cutaneous manifestations).

Treatment of DRESS syndrome requires a high-dose steroid regimen of at least 1 mg/kg/day of prednisone and a slow taper of six to eight weeks to prevent recurrence. In our case, the rapid tapering of prednisone resulted in a relapse. High potency topical steroids can be used for cutaneous involvement without organ involvement. Immunosuppressants such as cyclosporine and intravenous immunoglobulins are being studied for refractory cases. However, current data on their efficacy is limited and additional studies are required [12].

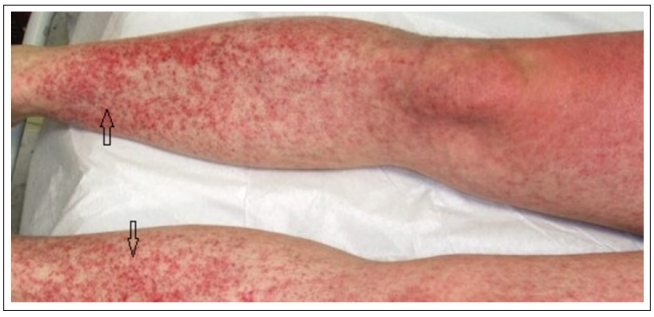

Figure 1: Lower extremities showing morbilliform, non-blanching rash (black arrows)

Conclusion

Physicians should be aware of clarithromycin and moxifloxacin as possible agents that can cause DRESS syndrome, a rare but life-threatening immune reaction. Patients should be educated on the need for a prolonged course of steroids and its associated adverse effects. Communication between caregivers about treatment schedules is vital in the transition of care. A close follow-up to monitor clinical improvement is necessary to prevent relapses, as in our case.

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