Japanese Journal of Gastroenterology and Hepatology

Research Article

ISSN: 2435-1210 | Volume 10

'DRESS' from a Gastroenterology & Hepatology Perspective

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Received: 08 Jan 2025

Accepted: 06 Feb 2025

Published: 12 Feb 2025

J Short Name: JJGH

Keywords:

DRESS; Adverse Drug Reaction; Rash(es); Eosinophilia; Transaminase

Citation:

Anu Jacob. 'DRESS' from a Gastroenterology & Hepatology Perspective. J Gastro Hepato. 2025; V10(14): 1-6

1. Abstract

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is an uncommon but potentially life-threatening form of adverse drug reaction (ADR), most frequently reported with antiepileptics and antibiotics. A multifactorial interplay involving genetic predisposition, impaired drug metabolism, and viral reactivation is proposed in the pathogenesis of DRESS syndrome. The constellation of rash, fever, lymphadenopathy, and eosinophilia with evidence of visceral involvement following drug initiation should alert the clinician of DRESS syndrome. The liver, pancreas, and gastrointestinal tract are common sites of visceral involvement in DRESS syndrome. Hepatic involvement, one of the most frequent manifestations, ranges from transient transaminitis to fulminant liver failure and often serves as a critical determinant of mortality. However, pancreatic and other gastrointestinal manifestations are often overlooked and underreported. Owing to the lack of definitive diagnostic tests, it depends on diagnostic criteria, preferably the RegiSCAR criteria. The lymphocyte transformation test, an in vitro technique, can be utilized to identify the culprit drug. Prompt diagnosis and withdrawal of the offending drug are the keys to management. Symptomatic and supportive measures and topical and systemic steroids often suffice the treatment, including for gastrointestinal and hepatobiliary manifestations. Acute liver failure is an indication of liver transplantation. Emerging insights into the pathophysiology, including genetic predispositions and viral reactivations, highlight the need for stratified diagnostic and therapeutic approaches.

2. Introduction

DRESS syndrome is a severe form of ADR, potentially life-threatening, with an estimated mortality ranging from 3.8% to 10%. [1,2] The incidence varies highly across published literature, predictably related to the drugs used, accuracy in diagnosis and reporting, and genetic risk factors; ranges from 1 in 10,000 to 1 in 100,000. [1,3]. The earliest, though vague, description of similar drug reactions dates to the era of hydantoin, a first-generation antiepileptic agent. With time, the syndrome got better defined and has been linked with an increasing number of molecules. In 1996, Bocquet et al. described the "drug rash with eosinophilia and systemic symptoms," but being a multisystem ADR, not limited and sometimes sparing the skin, the 'R' in the acronym DRESS was changed from 'Rash' to 'Reaction.' [1,4]. Drug-Induced Hypersensitivity Syndrome (DIHS), used and defined by the Japanese, is synonymous with DRESS syndrome[1,5].

3. Etiopathogenesis

Ever since the initial reports of hydantoin, an increasing number of medications have been implicated in DRESS syndrome. Currently, about 50 drugs have been linked with DRESS syndrome to date, increasing with time [3,6]. A 2011 systematic review by Cacoub et al. [7].Identified a causal association between 44 drugs and DRESS syndrome; the most frequently implicated were allopurinol and carbamazepine [7]. This complex ADR is also described with other antiepileptics, anti-infectives, sulfasalazine, omeprazole, olanzapine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, and more (Table 1) [1,7,8]. Amoxicillin is specially mentioned as it is

often considered an aggravating factor and less frequently causes DRESS syndrome by itself [1,9]. In 10-12% of cases of DRESS syndrome, the implicated drug remains unidentifiable. DRESS syndrome is more frequently reported among women and the black population; reasons unknown [3]. The exact pathogenesis of DRESS syndrome remains unclear; the hypothesized mechanisms include genetic predisposition, defects in drug metabolism, or reactivation of human herpes viruses (HHV), culminating in an immune-mediated response. Specific HLA haplotypes have been linked with DRESS syndrome and drugs: HLA-B*5701 (abacavir), HLA-DR3, HLA-DQ2, HLA-A*3101 (carbamazepine), HLA-B*5801 (allopurinol). Defective drug metabolism is best defined in the causality of DRESS syndrome related to antiepileptics. Deficient or defective enzymes, epoxide hydroxylase or glutathione transferase, accumulate toxic metabolites of antiepileptics and oxidative stress, with subsequent cellular toxicity and heightened inflammatory responses. HHV-6 has been identified in blood in 60-80% of patients with DRESS syndrome. Other herpes viruses, Epstein–Barr virus, cytomegalovirus, and HHV-7, have also been linked. The HHV-6, following the initial infection in early childhood, remains latent in the T cells. A reactivation of the virus, induced by the drug or as a bystander effect, eliciting an immune response is a postulated mechanism in DRESS syndrome. The association between HHV-6 and DRESS syndrome is well explained and included in the Japanese diagnostic criteria for DIHS[1,3,10-12].

Table 1: Drugs implicated in DRESS syndrome.

Antiepileptics	Phenytoin*, Lamotrigine*, Carbamazepine*, Phenobarbital*, Levetiracetam*, Valproate, Zonisamide
Antibiotics	Co-trimoxazole*, Dapsone*, Benznidazole, Vancomycin, Sulfonamides, Penicillins (esp. Piperacillin-Tazobactam*), Cephalosporins, Levofloxacin, Rifampicin*, Isoniazid, Pyrazinamide, Ethambutol,
Antivirals	Abacavir*, Nevirapine*, Telaprevir, Boceprevir
Others	Allopurinol*, Sulfasalazine*, Salazosulfapyridine*, Quinine, Iodinated contrast, Ranitidine, Imatinib, Leflunomide, Captopril, Aspirin, Ibuprofen. Celecoxib, Diltiazem.
** Frequent culprit drugs*	

4. Clinical Manifestations

4.1. General

The DRESS syndrome typically manifests within 2-6 weeks after initiating the offending drug; however, it may develop swifter following re-exposure. Fever is typically the earliest manifestation, seen in >90% of cases, shortly followed by skin rashes [1,13]. Erythematous pruritic morbilliform rashes spread rapidly from the face and upper trunk to the upper, followed by the lower extremities. Vesicular, target, or purpuric lesions may be seen less frequently. Rashes often involve >50% of body surface area, and around 50% have concomitant mucosal lesions. Generalized or localized lymphadenopathy occurs in as high as 75% of cases [14]. The triad of fever, rashes, and adenopathy may be followed by derangements of internal organs, most frequently hematologic and hepatic, and less frequently renal, pulmonary, cardiac, gastrointestinal, and neurologic involvements and manifestations. [1,3,14]. Hematological manifestations include leucocytosis, eosinophilia, leukopenia, thrombocytopenia, or anemia. Eosinophilia is frequent and is considered a diagnostic criterion for DRESS syndrome[3]. The hepatic manifestations may range from asymptomatic transaminase elevation to rare fulminant liver failure. The liver and gastrointestinal manifestations are further detailed in the subsequent sections [1,15]. Renal involvement is seen in 10-30% of cases, most frequently with allopurinol, carbamazepine, or dapsone-induced DRESS syndrome; it manifests as asymptomatic hematuria, proteinuria, and mild renal failure [1,3]. Pulmonary manifestations are most frequently reported with minocycline-induced DRESS syndrome, which manifests as pneumonitis, pleuritis, or rarely respiratory distress syndrome [1,3].

4.2. Hepatobiliary

The liver is amongst the most frequently involved organ systems in DRESS, likely next only to skin and hematologic manifestations. In a retrospective study, Chen et al. reported hepatic involvement in 80% of cases. [16]. Hepatic involvement may range from transaminitis, the most frequent, to fulminant liver failure, which is rare but happens to be the most frequent cause of mortality in DRESS syndrome. Hepatic dysfunction may precede other manifestations and follow a hepatocellular, cholestatic, or mixed pattern. Studies have variably reported hepatocellular and cholestatic patterns more frequently than the other; however, the Spanish DRESS guidelines state cholestatic patterns predominate [8,17,18]. Furthermore, it has been assessed that the hepatic dysfunction in DRESS follows a protracted course compared to other forms of drug hypersensitivity and could last even months after the resolution of skin lesions. Also, hepatic involvements correlated with higher rates of renal dysfunction and

mortality [19]. Wang et al. reported transaminitis exceeding 100 IU/L, hyperbilirubinemia, and liver failure in approximately 50%, 31%, and 23%, respectively, of Lamotrigine-associated DRESS [20]. Histological analysis of the liver demonstrated dense inflammatory infiltrates, comprising lymphocytes and eosinophils, together with hepatocyte necrosis [21]. The Spanish guidelines recommend the International Drug-Induced Liver Injury (DILI) Expert Working Group criteria for diagnosing and assessing liver injury severity[8]. Accordingly, DILI is diagnosed when the ALT exceeds ≥5 times the upper limit of normal (ULN), when the ALP exceeds ≥2 times ULN, or with a combination of ALT ≥3 times ULN plus total bilirubin ≥2 times ULN with normal ALP; all criteria apply when other causes of liver diseases have been excluded. The severity grading is summarized in Table 2. [8,22]. Hepatic encephalopathy, coagulopathy, and a low factor V to <40% level on 2nd day were reported as markers of poor outcome or liver transplantation [13,24]. Published literature reveals that sulfasalazine is most frequently associated with severe hepatic dysfunction and liver failure [25].Isolated biliary involvement is exceedingly rare in DRESS syndrome, though patients may have cholestatic hepatitis. Cases of vanishing bile duct syndrome associated with DRESS have been reported with Lamotrigine [26].

Table 2: DILI - diagnostic criteria and severity staging.

DILI diagnosis		DILI Severity
ALT ≥5x ULN, or	Mild	ALT or ALP meets the criteria, but TB <2x ULN
ALT >3x ULN + TB >2x ULN, or	Moderate	ALT or ALP meets the criteria, but TB ≥2x ULN
ALP ≥2x ULN, and	Severe	Moderate + INR ≥1.5, or Ascites, or Encephalopathy, or other organ failure presumed due to DILI
Alternate causes excluded	Fatal	Death or Liver transplantation

ALT: Alanine aminotransferase; ALP: Alkaline phosphatase, TB: Total bilirubin, ULN: Upper limit of normal, INR: international normalized ratio

4.3. Pancreatic

Pancreatic involvement is estimated not to be uncommon in DRESS syndrome. In a systematic review, Jevtic et al. reported the pancreas as the second most frequent site of visceral involvement, next to the liver. Acute pancreatitis and new-onset type 1 diabetes mellitus (DM) are reported during the acute or early phase. Less commonly, chronic pancreatic insufficiency may develop as a late sequelae. The mean duration to onset of type 1 DM was estimated as 84.4 days. Lamotrigine was reportedly the most frequent drug implicated in DRESS pancreatitis [27-31].

4.4. Gastrointestinal

The reported gastrointestinal symptoms in DRESS include diarrhea, abdominal pain, nausea, vomiting, and dysphagia, diarrhea being the most common. Colitis was the most frequently reported liminal manifestation; others include enteritis, esophagitis, and gastritis. Endoscopic evaluation in cases revealed inflammation, mucosal friability, and ulcerations. Tissue biopsies demonstrated lymphocytic infiltration with increased plasma cells and eosinophils [27]. Rare cases of gastrointestinal bleeding and bowel perforation have been reported in DRESS. [32-33] Adike et al. reported a case of autoimmune enteropathy in a patient with DRESS and protracted diarrhea. [34].

4.5. Diagnosis

There is no single diagnostic test or gold standard for diagnosing DRESS syndrome; instead, the diagnosis of DRESS syndrome relies on diagnostic criteria: at least three have been proposed and summarized in Table 3. Bocquet et al. proposed the earliest, while the European Registry of Severe Cutaneous Adverse Reaction (RegiSCAR) and Japanese group criteria were proposed later. RegiSCAR criteria are the most comprehensive and widely accepted, while the Bouquet et al. criteria are the simplest [35]. The Spanish guidelines recommend that DRESS syndrome be suspected in all individuals presenting with a constellation of skin rashes, fever, lymphadenopathy, eosinophilia, and atypical lymphocytes with evidence of hepatic or renal involvement, presenting within 2-8 weeks of initiation of a new drug. The guidelines recommend the RegiSCAR criteria for diagnosis of DRESS syndrome over other criteria [8]. The Spanish group recommends a battery of tests including but not limited to hemogram, renal and liver functions, inflammatory marker assay, polymerase chain reaction for herpes and hepatotropic viruses, parvovirus, antinuclear antibodies for diagnosis, severity assessment, and follow-up. The invitro Lymphocyte Transformation Test (LTT) may be utilized to identify the culprit drug. For the best results, LTT is recommended in the recovery phase of DRESS syndrome, preferably 6 -12 months after the reaction. The test is not to be performed during the first 4-8 weeks of the reaction while on or within 4 weeks of corticosteroid treatment. LTT has a 73% sensitivity and 82% specificity during the recovery phase of DRESS syndrome[8,36].

Table 3: Diagnostic criteria for DRESS syndrome.

RegiSCAR criteria				
Features		No	Yes	Unknown
Fever >38.5°C		-1	0	-1
Lymphadenopathy (>1cm, >2 sites)		0	1	0
Atypical lymphocytes		0	1	0
Eosinophilia	700-1499 cells/mm^3 or 10%-19.9%	0	1	
	≥1500 cells/mm^3 or ≥ 20%		2	
Skin rashes	Extent >50%	0	1	
	At least 2 of edema/infiltration/purpura/scaling	-1	1	
	Biopsy suggestive of DRESS	-1	0	
Internal organ involvement	One	0	1	0
	Two/more		2	
Resolving in ≥15 days		-1	0	-1
Exclusion of alternate causes		0	1	0
RegiSCAR score: <2: no DRESS; 2-3: Possible DRESS; 4-5: Probable DRESS; >5 Definite DRESS				

Japanese DIHS criteria		Bocquet et al. criteria
1. Maculopapular rash in 3 weeks of starting a new drug		1. Cutaneous drug eruption
2. Symptoms ≥ 2 weeks after discontinuation of the implicated drug		
3. Fever; >30°C		2. Hematological abnormalities Eosinophilia (>1.5 x 10^9/L), or Atypical lymphocytosis
4. Liver abnormalities (ALT >100 IU/L)		
5. Leukocyte alterations	Leukocytosis (>11 x 10^9/L)	
	Eosinophilia (>1.5 x 10^9/L)	3. Lymphadenopathy (>2cm), or Hepatitis (ALT > 2x ULN) Interstitial nephritis Interstitial pneumonitis Carditis
	Atypical lymphocytosis (>5%)	
6. Lymphadenopathy		
7. HHV-6 reactivation		
DIHS: all 7 criteria met. Atypical DIHS 5 of 7 criteria met		Diagnosis requires 1 + 2 + 3

ALT: Alanine aminotransferase; ULN: upper limit of normal; DIHS: Drug-Induced Hypersensitivity Syndrome, HHV-6: Human Herpes Virus-6

4.6. Management

The key to management is the prompt withdrawal of the offending drug or drugs. Patients should preferably be hospitalized and managed by a multidisciplinary team. Empirical use of antibiotics and non-steroidal anti-inflammatory drugs should be avoided, while hydration and electrolyte homeostasis should be ensured. In case of mild disease with no or mild organ involvement (stage 1 drug-induced liver injury or acute kidney injury), high-potency topical steroids for 1 week, close follow-up changes in severity, and supportive care will suffice. In more severe DILI, an intensive, multidisciplinary level of care with systemic steroids will be required in most cases. Oral prednisone, 0.8-1 mg/kg/day till 2-3 weeks or clinical improvement, is suggested, followed by a gradual taper of 5-10mg/week. In case of inadequate response with systemic steroids, other options include cyclosporine, intravenous immunoglobulin, cyclophosphamide, and plasmapheresis. The recommended dose of cyclosporine is 4-5mg/kg/day for 5-7 days, followed by 50mg/week taper. The dose for intravenous immunoglobulin is 2gm/kg, divided over 5 days. There is limited data on intravenous ganciclovir, 5mg/kg, or oral valganciclovir, 900mg twice daily, in severe cases wherein viral reactivation has been demonstrated or is strongly suspected[8].

The management of gastrointestinal manifestations of DRESS follows the principles mentioned earlier. As indicated, supportive and symptomatic measures include hydration, antiemetics, and antidiarrheals. In significant liver injury, the Spanish guidelines recommend oral methylprednisolone, 60-120 mg/day, or oral prednisone, 40-60mg/day initially, followed by steroid taper[8]. In case of acute liver failure, the only effective treatment is liver transplantation.

5. Conclusions

DRESS syndrome is a complex, multifaceted adverse drug reaction with significant implications for gastroenterology and hepatology. The liver is the most frequent site of visceral injury, and hepatic manifestations may range from asymptomatic transaminsemia or cholestasis to fulminant liver failure, which is regarded as the most frequent cause of mortality in DRESS. Acute pancreatitis, new-onset type 1 DM, colitis, enteritis, and gastritis manifesting as diarrhea, vomiting, and gastrointestinal bleeding are reported as well. Improved awareness about this rare syndrome and a high index of suspicion should improve the diagnostic yield. Effective management requires prompt identification and withdrawal of the offending drug, with

systemic corticosteroids playing a pivotal role in mitigating severe organ dysfunction. Further research into hepatobiliary-specific mechanisms and targeted therapies is essential to optimize patient outcomes and reduce the burden of this potentially fatal condition.

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