Arch Med Case Rep. 2026;8(1):5–10.

Archives of Medical Case Reports | Case Report

Vancomycin-Induced DRESS Syndrome with Nephritis, Pneumonitis, and Colitis in an HLA-A*32:01-Positive Patient: A Case Report

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Received date: December 09, 2025, **Accepted date:** January 28, 2026

Citation: Symes FLI, Bakthavatsalam K. Vancomycin-Induced DRESS Syndrome with Nephritis, Pneumonitis, and Colitis in an HLA-A*32:01-Positive Patient: A Case Report. Arch Med Case Rep. 2026;8(1):5–10.

Abstract

The diagnosis of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is challenging due to its variable clinical presentation and long latency period. We report a case of a woman in her 60s who developed Vancomycin-induced DRESS after treatment for cellulitis and septic arthritis. The patient presented with cutaneous, renal, pulmonary, and gastrointestinal involvement, but notably without hepatic injury. HLA testing confirmed HLA-A*32:01 positivity which suggested Vancomycin-induced DRESS. Withdrawal of Vancomycin and initiation of corticosteroids led to clinical improvement. However, a relapse occurred during the steroid tapering phase. Cyclosporine was commenced as a second line agent, which led to sustained remission. This case highlights the diagnostic and therapeutic challenges of DRESS, the potential for atypical organ involvement, HLA testing in determining pharmacogenetic susceptibility, and the importance of multidisciplinary input to optimize the care of the patient. Clinicians should consider DRESS in patients presenting with cutaneous and systemic symptoms after drug therapy, maintain vigilance for relapses during corticosteroid tapering, and recognize the utility of Cyclosporine in steroid-dependent or refractory cases.

Keywords: Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), Drug hypersensitivity, Vancomycin, HLA-A*32:01, Pharmacogenetics

Introduction

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a drug-induced hypersensitivity reaction that often manifests as a morbilliform rash and may involve multiple organs, most commonly the liver. Mortality approaches 10%, emphasizing the importance of early recognition and prompt withdrawal of the offending drug [1]. Its pathophysiology is incompletely understood but is likely multifactorial, involving a genetic deficiency in drug metabolism leading to toxic metabolite accumulation, specific human leukocyte antigen (HLA)- linked drug hypersensitivity, and viral reactivation that amplifies immune dysfunction [2]. Diagnosis is clinical, supported by the European Registry of Severe Cutaneous Adverse Reactions (RegiSCAR) scoring system [2,3], however early identification is difficult due to variable clinical presentation, long latency phase, and the need to rule out other systemic disorders. We present a case of Vancomycin-induced DRESS in a patient with confirmed HLA-A*32:01, highlighting the challenges in diagnosis, the role of pharmacogenetics, and the therapeutic role of Cyclosporine in steroid-dependent relapses.

Case Presentation

This case involved a female in her 60s who presented with a 2 day history of right lower limb pain and swelling. The clinical course is summarised in **Table 1**. She had a past medical history significant for recurrent lower limb cellulitis, chronic venous ulcers, seasonal asthma and sinusitis. She has an allergy to Penicillin (anaphylaxis). On examination, she had extensive cellulitis of her right lower limb, infected venous ulcers, and an irritable right ankle joint suspicious for septic arthritis. A previous wound swab grew Methicillin Resistant *Staphylococcus Aureus* (MRSA), hence she was commenced on intravenous Vancomycin. Ankle aspirate culture was later positive for Group C *Streptococcus*. Infectious diseases team planned a 4 week course of Vancomycin for septic arthritis.

Table 1. Timeline of clinical course.

Timeline	Key events
Day 0	• Hospital Admission • **Commenced on Vancomycin** for right lower limb cellulitis and right ankle septic arthritis.
Day 17	• Commenced on Moxifloxacin for hospital acquired pneumonia.
Day 21	• Commenced on 25 mg oral Prednisolone with a gradual taper for infective exacerbation of asthma, tested positive for Metapneumovirus.
Day 27	• **Onset of DRESS symptoms**; morbilliform rash and fevers, ongoing dyspnea. • Beginning of upward trend in eosinophils, creatinine, and inflammatory markers. • Dermatology review: likely a morbilliform drug exanthem secondary to Moxifloxacin. DRESS less likely.
Day 28	• **Vancomycin ceased**. • Clindamycin commenced for right ankle septic arthritis.
Day 34-37	• Peak creatinine and eosinophil levels. Onset of diarrhea. • Skin biopsy and HLA-A*32:01 test performed. • **Prednisolone increased from 15 mg to 80 mg daily**, with a gradual taper. • Co-trimoxazole commenced for PJP prophylaxis.
Day 41	• CT Chest: pneumonitis. • Skin biopsy results suggestive of a drug reaction. • Clinical and biochemical improvement.
Day 49	• HLA-A*32:01 positive, suggesting Vancomycin-induced DRESS.
Day 50	• **DRESS syndrome relapse;** recurrence of rash associated with eosinophilia • **Prednisolone increased from 50 to 60 mg daily, with a slower taper planned. Subsequently commenced on Cyclosporine.** • Co-trimoxazole ceased, Atovaquone commenced for PJP prophylaxis.

Abbreviations: DRESS: Drug Reaction with Eosinophilia and Systemic Symptoms; HLA: Human Leukocyte Antigen; PJP: *Pneumocystis Jirovecii* Pneumonia; CT: Computed tomography

She clinically improved with the above antibiotic regime. On day 17, she had likely hospital acquired pneumonia, and was treated with Moxifloxacin. On day 21, she was commenced on 25 mg Prednisolone daily for infective exacerbation of Asthma associated with positive Metapneumovirus. On day 27, she developed pruritic morbilliform erythema on her right lower limb which progressed to involve the entire body, and fevers. She subsequently developed diarrhoea, and had ongoing dyspnea. She was reviewed by Dermatology and further investigations were performed.

Investigations

Blood tests showed rising eosinophils (baseline 0.0 × 10^9/L, peak 2.8 × 10^9/L) and creatinine (baseline 73 umol/L, peak 302 umol/L), whilst liver function tests remained unremarkable. She had raised inflammatory markers associated with neutrophilia and lymphocytosis. Extensive investigations for infectious and autoimmune etiologies were negative, including that for Herpesviridae viruses. Urinalysis showed microalbuminuria and microscopic hematuria, but no casts, dysmorphic red cells or eosinophils. Stool cultures were negative, although faecal calprotectin was elevated (867 ug/g).

Computed tomography (CT) kidneys, ureters and bladders was unremarkable. High resolution CT chest showed ground-glass nodules with predominant centrilobular distribution, and peribronchial thickening with upper lobe predominance, suggestive of pneumonitis. Skin biopsy showed mild to moderate spongiosis in the epidermis, exocytosis with mild basal layer damage, superficial dermis had a mixed inflammatory cell infiltrate (with majority lymphocytes and a small number of eosinophils and neutrophils), suggestive of a drug reaction/DRESS. No vasculitis was seen, and immunofluorescent staining for IgG, IgM, IgA, C3, C1q and fibrinogen was negative. The patient tested positive for HLA-A*32:01, which is strongly associated with Vancomycin associated DRESS.

According to the European Registry of Severe Cutaneous Adverse Reactions (RegiSCAR) scoring system [2,3], the patient scored 7 points, categorizing the probability of Vancomycin induced DRESS syndrome as "Definite".

Differential diagnosis

On initial review by Dermatology, a morbilliform drug exanthem with eosinophilia secondary to a fluoroquinolone (Moxifloxacin) commenced 10 days earlier was deemed the most likely diagnosis. With progression of the rash associated with rising eosinophilia and creatinine, DRESS was considered a possible differential diagnosis. However, given that the patient had a 1-week course of Vancomycin 4 years ago with no adverse reactions, this episode would be considered a rechallenge, hence DRESS onset would typically be quicker than the expected two to eight weeks. Another atypical feature was the lack of liver involvement. Given the temporal relationship with Moxifloxacin, a morbilliform drug exanthem secondary to Moxifloxacin was initially believed to be more likely. Eosinophilic granulomatosis with polyangiitis (EGPA) was also considered, however the Rheumatology team deemed it unlikely.

Subsequently, the presence of widespread morbilliform rash, multiorgan involvement (renal, pulmonary and gastrointestinal), eosinophilia, characteristic skin biopsy findings, positive HLA-A*32:01, and a RegiSCAR score of 7 confirmed a diagnosis of Vancomycin-induced DRESS.

Treatment and outcome

Vancomycin was ceased on day 28 and the patient was commenced on 80 mg of Prednisolone daily (0.6 mg/kg) on day 37, which was gradually tapered to 50 mg daily. Co-trimoxazole was commenced for Pneumocystis jirovecii pneumonia (PJP) prophylaxis. The rash significantly improved and eosinophil count normalized within a week. There was a more gradual improvement in renal function over the next 2 months.

On day 50, the patient had a recurrence of cutaneous rash associated with eosinophilia whilst on 50 mg of oral Prednisolone daily. It was most likely a relapse of DRESS triggered by tapering steroids too rapidly. The Prednisolone dose was increased to 60 mg with a slower taper, and the patient clinically and biochemically improved. Co-trimoxazole was also ceased and Atovaquone was commenced for PJP prophylaxis. Repeat testing for reactivation of herpesviruses were negative. Due to an anticipated protracted course of high dose steroids with the patient already exhibiting cushingoid features, Cyclosporine was commenced at 150 mg twice a day (2.5 mg/kg/day) as a steroid-sparing agent.

The patient tolerated Cyclosporine well, with stable renal function. Cutaneous, renal, pulmonary, and gastrointestinal manifestations gradually resolved. She received extensive allied health input to help improve her mobility and functional status, however due to ongoing high care needs, she was eventually discharged to a residential aged care facility.

Discussion

Complex interplay between drugs, viruses and the immune response

DRESS is associated with the reactivation of viruses from the Herpesviridae family [for example human herpesvirus (HHV) 6, HHV-7, cytomegalovirus (CMV), Epstein-Barr virus (EBV), [4] however the specific nature of the association has not been clarified. It is hypothesized that drug-specific immune responses induce viral reactivation in latently infected cells [5]. There has been little data on whether acutely acquired viral infections such as respiratory viruses could possibly be associated with DRESS.

Our patient tested positive to Metapneumovirus 1 week prior to the onset of morbilliform rash and fevers (day 27). She also tested positive to Influenza A 1 week prior to the relapse of DRESS syndrome on day 50. This could be a purely incidental finding, however there have been case reports of drug-induced DRESS associated with respiratory infections. Shalom *et al.* reported a case of DRESS secondary to Cephalosporins associated with *Mycoplasma pneumoniae* infection, in the context of a known penicillin allergy. It was hypothesized that the mycoplasma infection may have lowered the drug allergy threshold, or there could be possible synergistic effects between an acute viral infection and a known drug allergy, triggering a DRESS eruption [6]. There has also been a case of DRESS secondary to Sulfasalazine associated with positive serology for influenza A and B [7], and a few cases of DRESS associated with influenza vaccinations [8–10]. It was hypothesized that vaccines which act as immune stimulants could potentially trigger a non-specific immune activation that results in DRESS [8,10].

In our patient, in addition to drug hypersensitivity, an anti-viral immune response may have contributed to a severe and prolonged course of DRESS, and a relapse. Nevertheless, we note that eosinophilic drug reactions following acute respiratory viral infections are rare.

Drug hypersensitivity to previously tolerated Vancomycin

DRESS syndrome is a T-cell mediated type four hypersensitivity reaction, that has a typical latency period of between 2 and 8 weeks before the onset of symptoms. The latency period may differ for different medications [11] Re-exposure after prior asymptomatic sensitization usually results in a more rapid onset of DRESS eruption [12–14] Our patient developed DRESS only after a prolonged 4 week course of Vancomycin despite prior sensitization (The patient tolerated a 1 week course of Vancomycin 4 years ago). This could be due to several factors. Prednisolone given for the presumed asthma exacerbation could have dampened the immune response. The risk of

DRESS may also be impacted by drug dosage and duration, delayed drug clearance and accumulation [15–17] This could possibly explain why the patient experienced Vancomycin induced DRESS only after an extended exposure.

A hypersensitivity reaction to another drug (such as Moxifloxacin) could have triggered new sensitization to a previously well tolerated drug [18,19]. However given that our patient had pharmacogenetic susceptibility to Vancomycin induced DRESS with a positive HLA-A*32:01, [20, 21] it is likely that the patient intrinsically had a higher risk of hypersensitivity to Vancomycin.

Vancomycin-induced nephrotoxicity and acute interstitial nephritis

Vancomycin-induced nephrotoxicity is reported in up to one fifth of patients receiving Vancomycin therapy, with the most likely mechanism being increased oxidative stress in the renal tubules resulting in acute kidney injury [22]. In a separate mechanism, Vancomycin-induced acute interstitial nephritis (AIN) involves a type IV hypersensitivity to a drug and is characterized by immune-mediated tubulointerstitial damage [23–25]. In a retrospective study by Madigan *et al.*, there was a higher incidence of renal injury in Vancomycin associated DRESS (75% in the case series and 68% in a literature review), [24] as compared to the overall incidence in DRESS (12–40%) [3,26,27].

Although the mechanism of renal injury in our patient remains uncertain without a renal biopsy, the timing and clinical response to drug withdrawal and steroid treatment are suggestive of acute interstitial nephritis occurring as a feature of DRESS.

DRESS relapse

On day 50, the patient had a recurrence of cutaneous rash associated with eosinophilia, and this flare of DRESS was likely triggered by a rapid tapering of corticosteroids. Relapses have also been known to be triggered by medications which may be structurally different from the culprit drug [28–30]. It is hypothesized that the cross-reactivity to other drugs is due to immune hyperactivation that leads to polysensitization to multiple drugs [28–30]. Hence it is possible that the flare of DRESS could have been triggered by an unrelated drug, Co-trimoxazole, which was then ceased.

Cyclosporine

The first line treatment for DRESS is systemic glucocorticoids, and patients usually require a slow taper of 3 to 6 months to minimize risks of relapses or flare ups. However, glucocorticoids administered at high doses for a prolonged period of time can result in significant adverse effects such as hypertension, peptic ulcers, hyperglycemia and insulin resistance, adrenal insufficiency, and immunosuppression leading to a higher risk of infections.

Cyclosporine is a calcineurin inhibitor, and has been used as a second line treatment in corticosteroid-dependent DRESS [31–34]. In a retrospective review of a case series with 8 patients who had suboptimal response to a 3 week course of systemic corticosteroids and at least 1 relapse whilst tapering systemic corticosteroids, Cyclosporine was commenced with either discontinuation or tapering of steroid [31]. All patients had significant improvement in cutaneous symptoms and biochemical markers, had no further relapses, and experienced no serious adverse effects from Cyclosporine [31].

Our patient was commenced on Cyclosporine 150 mg BD (2.5 mg/kg/day) and the steroid was tapered. There was good response clinically, her renal function remained stable, and there were no further flares of DRESS.

Conclusion

We present a case of Vancomycin-induced DRESS syndrome with some atypical characteristics. It was unexpected to have a 4 week latency period prior to the onset of symptoms despite previous sensitization to Vancomycin. The patient also had no liver involvement even though it is most commonly affected in DRESS. Instead, the patient exhibited nephritis, pneumonitis and colitis, a combination that is rare with only one reported case in literature to the best of the author's knowledge [35]. The patient had a flare with tapering of steroids and thus required a second line agent Cyclosporine. Increased awareness of atypical manifestations and clinical course of DRESS will help guide clinicians in diagnosing future cases in a timely manner and optimizing treatment for patients.

Learning Points

The diagnosis of DRESS, particularly in the early stages, remains challenging due to its latency period and variable clinical presentation. Although the liver is the most commonly involved organ in DRESS, there is a subset of patients who do not exhibit any liver manifestations. For example, our patient had nephritis, pneumonitis and colitis, but no hepatitis.

Despite prior sensitization, a rechallenge may not necessarily result in a more rapid onset of DRESS as this may be influenced by other factors such as the drug class, dosage, duration and clearance. DRESS may also occur in response to previously tolerated drugs due to a hypothesized cross-reactivity to other drugs in the context of immune hyperactivation.

Human leukocyte antigen testing is helpful in determining the pharmacogenetic susceptibility of DRESS syndrome in particular populations to specific medications. In this case it provided supporting evidence that our patient, a European with positive HLA-A*32-01 likely had Vancomycin-induced DRESS.

Systemic steroids is the first line therapy for DRESS, whilst second line therapies include other immunosuppressive agents such as Cyclosporine. DRESS flare-ups are common and can be caused by several reasons including reactivation of herpesviridae viruses, rapid tapering of steroids, or triggered by other medications.

This case highlights the diagnostic challenges in the context of a complicated hospital admission with multiple medications being commenced, and emphasizes the importance of multidisciplinary input to optimize the care of the patient.

Conflicts of Interest

The authors declare no conflicts of interest.

Funding Statement

This case report did not receive any funding.

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