

Similar erythematous, flagellate eruptions have been observed in response to bleomycin treatment and dermatomyositis; however, these presentations often have mucous membrane involvement, which is not seen with shiitake-induced flagellate dermatitis.⁵ In addition, lesions resulting from bleomycin tend to take on a more hyperpigmented appearance. Another challenge in the diagnosis of shiitake dermatitis is the variable time course of eruption, which has been documented to appear anywhere from 12 hours to several days after mushroom consumption.

Susceptibility to shiitake dermatitis is variable, with an estimated prevalence of approximately 2%.⁶ Further studies must be performed to reflect the growing shiitake mushroom market and resultant exposure. The popularity of shiitake mushrooms in the United States has increased dramatically in recent years, as Asian cuisine has become a staple of the American culinary landscape.⁷ As this mushroom continues to integrate into the Asian-American palate, it is likely that physicians will see more similar cases. Such a trend calls for increased documentation and discussion of this relatively obscure condition.

Serena Fang, BA*
Amir Bajoghli, MD†
Mehdi Bajoghli, MD‡

*University of Utah School of Medicine
Salt Lake City, Utah

†Virginia Commonwealth University School of Medicine
Inova Health System
McLean, Virginia

‡Georgetown University School of Medicine
McLean, Virginia
serena.b.fang@gmail.com

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DRESS syndrome associated with splenic thrombosis



Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is a rare and severe hypersensitivity reaction that can occur after exposure to different medications. Onset occurs 2 to 8 weeks after drug exposure and the course can be prolonged with waxing and waning of symptoms despite withdrawal of the offending agent. Rash, facial swelling, lymphadenopathy, peripheral eosinophilia, and transaminitis classically characterize the reaction. However, different symptoms are reported, including numerous hematologic abnormalities and multiorgan involvement. Thrombotic complications are a known complication of hypereosinophilic conditions but are not well described in DRESS syndrome. We present a case of a 27-year-old African American woman with a medical history of mild intermittent asthma, acne, iron deficiency anemia, and hidradenitis suppurativa. She presented to our emergency department with nausea, abdominal pain, lightheadedness, fatigue, fevers, diffuse lymphadenopathy, facial edema, and loss of appetite after 4 weeks of treatment with minocycline and rifampin for a hidradenitis flare. Her history was notable for recent travel to the Middle East, Asia, and Europe and absence of personal or family history of clots. At initial evaluation she was found to be febrile (102.2°F) with high-normal blood pressure (132/88 mm Hg) and heart rate (92 beats/min); her respiratory rate and oxygen saturation were normal. Her laboratory results at presentation showed anemia with mild thrombocytopenia and mild leukocytosis (hemoglobin 6.8 g/dL [11.2–15.7 g/dL], hematocrit 28.0% [34–45%], platelets 128 K/ μ L [150–400 K/ μ L], white blood cell count 10.9 K/ μ L [4.0–10.0 K/ μ L]). Her differential was significant for 13% atypical lymphocytes and increased absolute eosinophil and lymphocyte counts (0.76 K/ μ L [range, 0.04–0.54] and 4.47 K/ μ L [1.2–3.7 K/ μ L], respectively). Her red blood cell smear also was diffusely abnormal but without a predominant red blood cell type and notably there were no schistocytes at initial evaluation (she later had occasional +1 schistocytes similar to the rest of the smear). Her prothrombin time (14.3 seconds; range, 9.4–12.5 seconds) and international normalized ratio

(1.3 seconds; range, 0.9–1.1 seconds) were mildly increased as were her liver function test results (alanine aminotransferase, 53 IU/L [0–40 IU/L]; aspartate aminotransferase, 78 IU/L [0–40 IU/L]; alkaline phosphatase, 145 IU/L [35–105 IU/L]; lactate dehydrogenase, 338 IU/L [94–250 IU/L]) with a normal total bilirubin level (0.4 mg/dL [range, 0–1.5 mg/dL]). Unfortunately, her liver enzymes continued to increase, and on hospital day 2 computed tomography with contrast of the abdomen and pelvis showed splenomegaly with multiple peripheral wedge-shaped areas of splenic hypoattenuation consistent with splenic infarct (Fig 1), for which she was started on intravenous heparin. However, her absolute eosinophil count did not increase and her eosinophil percentage remained normal. Given her hepatic involvement, splenic infarction, and hematologic findings, she underwent an extensive autoimmune, infectious, hypercoagulable, and malignant workup. Laboratory investigation to determine a cause for her symptoms was entirely negative except for a weakly positive anti-smooth antibody (titer 1:40).

She continued to have fevers in the setting of negative blood cultures without a clear infectious source; thus, treatment with antibiotics was deferred. Five days after admission she developed a sudden marked eosinophilia to 3.27 K/ μ L (15%) with a diffuse erythematous maculopapular rash and worsening facial swelling. At the time of rash occurrence, her RegiSCAR group diagnosis score for DRESS was 7, suggesting a definite diagnosis of DRESS (cutoff points: <2, no DRESS; 2–3, possible DRESS; 4–5, likely DRESS; and $\geq$ 6, definite DRESS).¹ Given the likelihood of DRESS syndrome, skin biopsy was deferred and she was started on 100 mg/d of intravenous methylprednisolone, with rapid symptomatic improvement over 48 hours. In the subsequent 48 hours, she had almost total resolution of her rash with slight improvement in her eosinophilia (from 18% to 8%; absolute count, from 4.01 to 2.14 K/ μ L). Despite ongoing steroid therapy without change in dose, on hospital day 8, she experienced a worsening of her rash and eosinophilia. Her steroids were continued at the same dose and her liver enzymes peaked at an alanine transaminase level of 233 IU/L and an aspartate transaminase level of 242 IU/L on days 8 and 11, respectively.

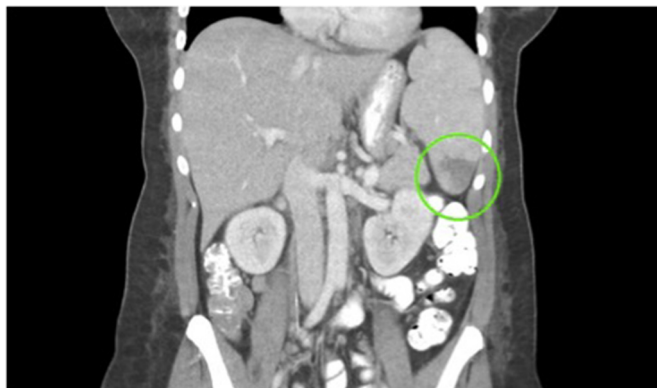


Figure 1. Computed tomogram with contrast depicting splenomegaly with multiple peripheral wedge-shaped areas of splenic hypoattenuation, consistent with splenic infarct.

Her eosinophilia peaked at 4.68 K/ μ L (31%) on hospital day 11, despite daily symptomatic improvement. On hospital day 13, after continued improvement, she was transitioned to apixaban and 100 mg/d of oral prednisone. She was discharged home on day 14 with close follow-up and a plan for slow steroid taper by 20 mg weekly for 2 weeks, followed by 5 to 10 mg weekly over 2 months. At her 2-week follow up with dermatology, she was on 60 mg/d of prednisone with resolution of her eosinophilia and normal liver enzymes. During her steroid taper to lower than 60 mg, she had a mild increase of her liver enzymes (alanine transaminase, 44–67 IU/L; aspartate transaminase, 41–49 IU/L) but remained asymptomatic without recurrence of her peripheral eosinophilia.

This patient's presentation and hepatosplenic involvement was initially concerning for infectious or inflammatory etiology, thus delaying a correct diagnosis until manifestation of her rash. DRESS associated with minocycline therapy is well documented; however, there are case reports of rifampin-induced DRESS, especially in the setting of tuberculosis treatment. Therefore, it is unclear in this case which medication was the culprit drug. A scoping review of the literature including PubMed, Google scholar, EMBASE, and Web of Science uncovered at least 10 cases of hypercoagulability associated with DRESS syndrome. Of these cases, 2 described deep venous thrombosis,^{2,3} 1 described hepatic thrombosis with necrosis,⁴ 1 described embolic stroke,⁵ and 1 described ventricular thrombus.⁶ The remaining 5 described systemic hypercoagulable pathology (thrombotic thrombocytopenic purpura,^{7,8} disseminated intravascular coagulation,^{9,10} and antiphospholipid syndrome¹¹). The causal agents in these reports were allopurinol,^{2,4,10} minocycline,^{3,6} phenytoin,^{7,8} dapsone,⁹ and sulfasalazine.¹¹ The pathophysiology

of hypercoagulability is unclear, but possible mechanisms include direct enhanced tissue factor expression by blood eosinophils, eosinophil inhibition of thrombomodulin function, and eosinophil-related endothelial damage activating the coagulation cascade.¹² Additional studies are needed to determine the risk of thrombosis in the setting of DRESS. However, these preliminary findings suggest that there could be an increased risk of thrombosis in patients with severe DRESS syndrome.

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Nicole LaHood, MD

Kristin Sokol, MD

Beth Israel Deaconess Medical Center

Boston, Massachusetts

nlahood@bidmc.harvard.edu

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Allergic sensitization in American children of Middle Eastern ethnicity at age 2



Children of Middle Eastern ethnicity (MEE) born in the United States are seldom specifically examined in allergic disease research, and the prevalence of allergic sensitization in these children is unknown.¹ For analyses, these children can be combined with white children not of MEE or excluded entirely because of the small sample, as we have previously done.^{2,3} However, this subgroup could have a unique set of cultural norms and/or

environmental exposures that distinguish them from white, black, non-Middle Eastern, and non-Hispanic children with respect to allergic disease risk factors.^{4,5} We previously observed disparities in allergic disease prevalence between 2-year-old black and white children in our unselected birth cohort study, the Wayne County Health, Environment, Allergy and Asthma Longitudinal Study (WHEALS), but had not investigated potential differences in MEE children.³

Our objective was to describe and compare allergic sensitization rates and total immunoglobulin E (IgE) levels between children who were identified as white, non-Middle Eastern, and non-

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