

Augmentin-Induced DRESS Syndrome with Interstitial Pneumonitis: A Rare Case Report

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Introduction

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a severe hypersensitivity reaction that typically presents with fever, rash, eosinophilia, and multi-organ involvement. Though primarily affecting the liver, kidneys, and hematologic system, pulmonary involvement is a rare but potentially life-threatening manifestation. Augmentin (amoxicillin-clavulanate) is a widely prescribed antibiotic, but its potential to cause DRESS with lung involvement is under recognized. Here, we have an interesting case of a 71-year-old woman who developed DRESS with pulmonary involvement following Augmentin therapy, emphasizing the importance of recognizing antibiotic-induced hypersensitivity syndromes.

Case Presentation

A 71-year-old woman with a history of immune thrombocytopenia, chronic lymphocytic leukemia (CLL), hypertension, hyperlipidemia, and biopsy-proven sarcoidosis presented with generalized weakness, dyspnea, fatigue, congestion, and persistent cough. She had completed a 10-day course of Augmentin for an ear infection but discontinued it early due to the development of a diffuse erythematous rash. She subsequently experienced decreased appetite, malaise, and progressive fatigue.

On admission, she was hypoxic with an oxygen saturation of 83% on room air, requiring supplemental oxygen via nasal cannula. Initial laboratory workup showed hyponatremia, but procalcitonin, proBNP, and troponin levels were negative, effectively ruling out bacterial infection and congestive heart failure (CHF).

A chest radiograph revealed diffuse reticular opacities. Chest CT demonstrated extensive bilateral ground-glass opacities with interlobular septal thickening, suggestive of an interstitial pneumonitis. Notably, no pleural effusion or pulmonary embolism was detected.

Despite negative blood, urine, AFB, fungal, and sputum cultures, as well as unremarkable TSH and cortisol levels, the patient's respiratory status deteriorated, necessitating increased oxygen support. A 2D echocardiogram showed a left ventricular ejection fraction (LVEF) of >70% with a hyperdynamic left ventricle and stage I diastolic dysfunction, essentially ruling out cardiogenic pulmonary edema.

She was promptly treated with high-dose systemic corticosteroids (IV Solu-Medrol 40 mg twice daily), along with bronchodilator therapy (DuoNeb, albuterol).

Subsequent chest CT examination performed 2 weeks after admission demonstrated significant worsening with bilateral consolidation, reticulation, and traction bronchiectasis in areas of previously seen ground glass opacity. Despite aggressive therapy with steroids and antibiotics patient's condition deteriorated and ultimately, she passed away within 2 weeks of hospital admission.

Discussion

Pulmonary involvement in DRESS syndrome is a rare and can be a serious complication. Imaging findings are non-specific with diagnosis is typically made on the basis of clinical history, physical examination, and bloodwork. Chest radiographic findings include reticulation, consolidation, pleural effusions, and lymphadenopathy. CT findings include ground glass opacities, consolidation, and nodules in addition to pleural effusions and lymphadenopathy. Distribution of imaging findings can be variable. While our case had findings in a predominantly central pattern, one case series reported a peripheral distribution as the most common.

Clinicians should maintain a high index of suspicion for medication-induced reactions in patients presenting with unexplained pulmonary findings following antibiotic use.

Conclusion

This case emphasizes the need for clinicians to consider drug-induced interstitial pneumonitis as a manifestation of DRESS syndrome. While Augmentin is commonly used, its potential for severe hypersensitivity reactions should not be

overlooked. Early diagnosis, drug discontinuation, and corticosteroid therapy are essential to improving patient outcomes.

References:

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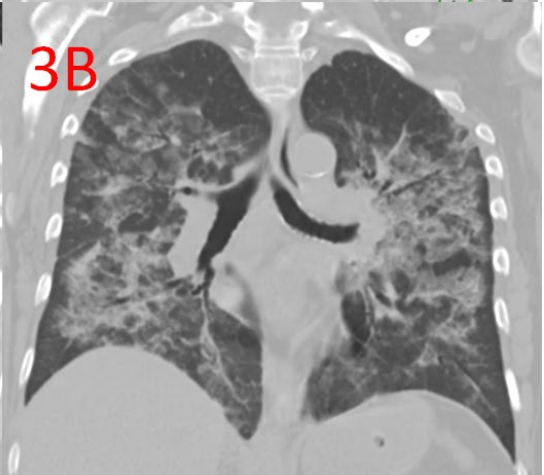
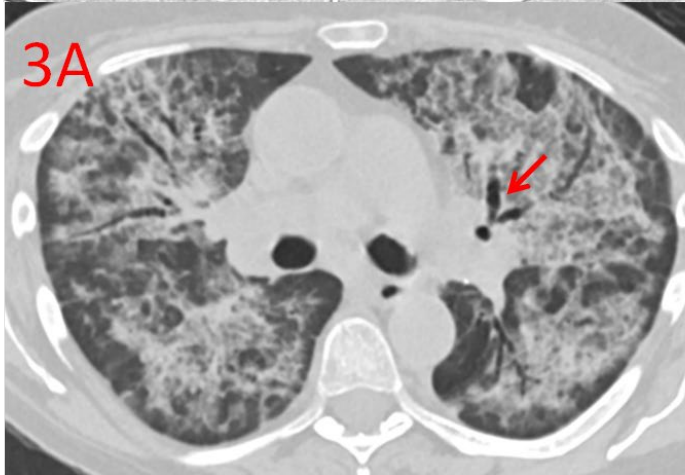
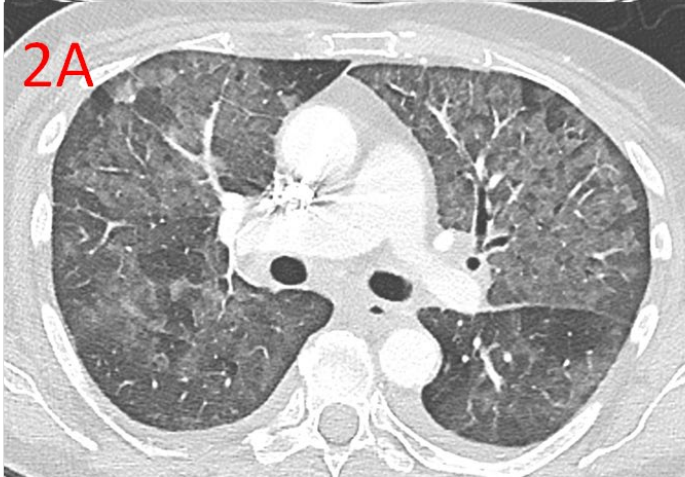
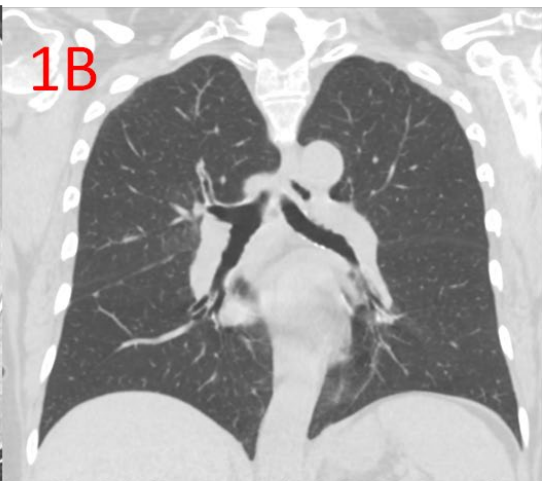
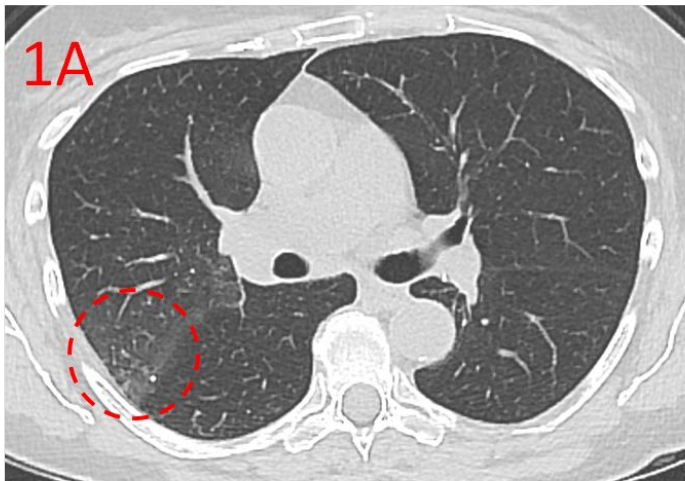
[F Skowron¹](#), [B Bensaid²](#), [B Balme³](#), [L Depaepe³](#), [J Kanitakis⁴](#), [A Nosbaum²](#), [D Maucort-Boulch^{5,6,7}](#), [F Bérard^{2,8}](#), [M D'Incan⁹](#), [S H Kardaun¹⁰](#), [J F Nicolas^{2,8}](#)

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Figs 1A and B: Baseline axial and coronal CT chest images 1 year prior to admission demonstrate relatively clear lungs with minimal clustered nodularity in the right upper lobe (dashed circle) likely reflecting an infectious/inflammatory bronchiolitis

Figs 2A and B: Axial and coronal CT chest images on admission demonstrate confluent peribronchial groundglass opacity throughout both lungs with mild superimposed interlobular septal thickening in keeping with pneumonitis.

Figs 3A and B: Axial and coronal CT chest images 2 weeks after admission demonstrate consolidation, reticulation, and traction bronchiectasis (arrow) throughout both lungs in areas of previously seen groundglass opacity suggesting organization and progression of diffuse alveolar damage.