Case Report

Rare Presentation of Ceftriaxone-Induced Hypersensitivity Pneumonitis

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Abstract

Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is a complex syndrome of varying intensity, clinical presentation, and natural history rather than a single, uniform disease. Numerous inciting agents have been described including, but not limited to, agricultural dusts, bioaerosols, microorganisms (fungal, bacterial, or protozoal), and certain drugs. Prompt diagnosis of HP is important, as the disease is reversible when diagnosed early in its course. Correct diagnosis is based upon exposure history, clinical assessment, radiographic and physiologic findings, and, if possible, the response to avoidance of the suspected etiologic agent.

Keywords: Ceftriaxone; Hypersensitivity pneumonitis; Drug-induced pneumonitis

Introduction

Drug-induced hypersensitivity pneumonitis is an immune response, normally associated with inhaled agents that can present with varying degrees of respiratory distress. It is important to recognize the effect of drug reaction on the body and differentiate from primary lung pathology. We present a case of hypersensitivity pneumonitis induced by a commonly administered medication, ceftriaxone [1]. This patient originally presented asymptomatic, with a rapid progression and then resolution of symptoms.

Case Report

This is a 60-year-old male, receiving treatment for right hallux osteomyelitis with ceftriaxone and vancomycin as an outpatient. On a routine follow-up visit, he was sent to the hospital after presenting with a fever, as well as an incidental finding of elevated creatinine. Upon admission, only vancomycin was discontinued, and admission chest X-ray (CXR) was within normal limits (Fig. 1). On admission day two, he developed acute respiratory failure requiring mechanical ventilation with CXR findings of right-sided infiltrate and non-specific left-sided changes (Fig. 2). No skin rash was noted and the labs did not show eosinophilia at the time. Bronchoalveolar lavage was negative for microorganisms, ruling out the differential of Aciinetobacter pneumonia. Ceftriaxone was empirically stopped, given the rare possibility of hypersensitivity pneumonitis. After cessation of ceftriaxone, the patient’s condition improved significantly and was extubated, CXR demonstrated improvement (Fig. 3).

Discussion

The original differential diagnosis for this case was broad, ranging from health-care-associated pneumonia to pulmonary embolism to drug-induced lung injury. The acute presentation, however, helped limit the differential, with interstitial pneumonitis being high on the list. Possible etiologies were ruled out by negative extremities ultrasound and bronchoalveolar

Figure 1. Chest X-ray at admission was “within normal limits”.

Manuscript submitted March 9, 2018, accepted April 17, 2018

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doi: https://doi.org/10.14740/jmc3046e
lavage negative for microorganisms. In this case, lack of rash
and eosinophilia lessened the possibility of a hypersensitiv-
ity reaction, though rapid improvement after cessation of the
causative agent was supportive of this diagnosis.

Interstitial pneumonitis is defined as an inflammation
change of the lung interstitium, including the alveolar septa,
due to a broad range of etiologies. The etiology of interstitial
pneumonitis can either be idiopathic or from known causes,
such as drug-induced [2, 3]. In immunocompromised popu-
lations, interstitial pneumonitis is more commonly caused
by fungal, atypical bacterial, and viral [4] infections [5]. In
healthy populations, the cause of interstitial pneumonitis is
more commonly an extrinsic particulate; fungal or plant an-
tigens in farmer’s lung [6], aerosolized metallic or organic
chemicals [7], or even possibly radiation [8].

The presentation of drug-induced pneumonitis commonly
includes dyspnea, fever, cough, and rash. Investigation will
often reveal inflammatory changes on CXR or CT [9], and eo-
sinophilia on lab analysis. If a drug hypersensitivity reaction is
suspected as the cause of pneumonitis, the medication can be
restarted after cessation to confirm the reappearance of symp-
toms; this however may cause the patient to require intubation
and supportive treatment. As with other hypersensitivity reac-
tions, the first step in treatment of drug-induced pneumonitis,
after life supporting measures, is cessation of the drug or in-
halation agent [10]. Patients with severe disease may require
treatment with corticosteroids [10], however, this has been
shown to have no effect on long-term outcomes [11].

Conclusions

While hypersensitivity reactions are a known adverse effect
of the use of cephalosporins, there are few reported cases of
pneumonitis associated with ceftriaxone [1]. This case is sug-
gestive that clinicians should continue to be aware of the possi-
bility of drug-induced pneumonitis, even in the absence of rash
or eosinophilia. At this time, more investigation is required to
establish the exact mechanism behind ceftriaxone-induced
pneumonitis, and to determine if there is population with an
elevated risk of this complication. We believe that the addition
of hypersensitivity pneumonitis to the differentials of patients
with similar presentations, and early recognition of this compi-
lcation can reduce or even prevent significant morbidity and
mortality.

References

1. Lee SH, Kim MH, Lee K, Jo EJ, Park HK. Hypersen-
sitivity pneumonitis caused by cephalosporins with
identical R1 side chains. Allergy Asthma Immunol Res.
2. King TE. Epidemiology and causes of hypersensitivity
pneumonitis. In: UpToDate, Post TW (Ed), UpToDate,
Waltham, MA. (Accessed on December 16, 2016.)
3. King TE. Diagnosis of hypersensitivity pneumonitis. In:
UpToDate, Post TW (Ed), UpToDate, Waltham, MA.
(Accessed on December 15, 2016.)
4. Travis WD, Fox CH, Devaney KO, Weiss LM, O’Leary
TJ, Ogniben FP, Suffredini AF, et al. Lymphoid pneumo-
nitis in 50 adult patients infected with the human immu-
nodeficiency virus: lymphocytic interstitial pneumonitis
versus nonspecific interstitial pneumonitis. Hum Pathol.
5. de Blic J, McKelvie P, Le Bourgeois M, Blanche S, Be-
oist MR, Scheinmann P. Value of bronchoalveolar lav-


