ejpmr, 2018,5(3), 143-144

SJIF Impact Factor 4.897

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case report

ISSN 2394-3211

EJPMR

A CASE OF SYSTEMIC HYPEREOSINOPHILIA ASSOCIATED WITH DOXYCYCLINE USE

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Article Received on 07/01/2018 **Article Revised on 28/01/2018** **Article Accepted on 17/02/2018**

ABSTRACT

Hypereosinophilic syndromes have been attributed to certain drugs in the literature, including minocycline. These syndromes may be restricted to a single organ or may involve multiple systems. We report the case of a 22-year-old woman with a history of atopy who developed peripheral hypereosinophilia after treatment with doxycycline for cellulitis, which was later found to be eosinophilic in nature. Doxycycline was discontinued on admission, but several days later she developed respiratory distress and bilateral infiltrates on chest radiograph. Bronchoalveolar lavage revealed 56% eosinophils consistent with eosinophilic pneumonia. To our knowledge, this is the first reported case of a hypereosinophilic syndrome following exposure to doxycycline.

KEYWORD: Hypereosinophilia, Bronchoalveolar, eosinophilic.

INTRODUCTION

Hypereosinophilic syndrome is a disorder of elevated peripheral eosinophilia (>1.5 x 10^9/L) with dysfunction of one or more organ systems. Among those systems often affected are dermatologic, pulmonary, and gastrointestinal. A number of etiologies have been identified, including helminth infections, iatrogenic, and idiopathic.

CASE REPORT

The patient is a 22-year-old woman with past medical history of asthma, eczema, and multiple allergies who presented initially with a diffuse urticarial rash that had first developed about one month before. Prior to admission, the patient had presented to an outside emergency room with a rash for which she was treated with clindamycin and Bactrim. She described the rash as "hives", which she had had many times in the past with no clear trigger. Routine labs drawn around that time indicated that the patient's eosinophils were at 3%. Four days later with worsening of the rash, she presented to an outpatient clinic where Bactrim was discontinued, and she was started on doxycycline. About one week after starting the doxycycline, the patient began to notice diffuse edema most pronounced in her extremities. Her peripheral eosinophil percentage had increased to 6% at that time. Two days later she was admitted to the hospital for diffuse worsening rash and edema. On the day of admission her peripheral eosinophils were at 11%. Doxycycline was discontinued on admission. Dermatology was consulted for assistance in identifying the rash. Skin biopsy was performed and revealed eosinophils and flame figures with no evidence of vasculitis, essentially ruling out a diagnosis of Churg-Strauss syndrome which had previously been part of the differential diagnosis. The patient was determined to have eosinophilic cellulitis (Well's syndrome) based on the results of the biopsy. Days after the biopsy, the patient suddenly developed respiratory distress and required transfer to the ICU. A diagnostic chest X-ray revealed acute, severe pulmonary edema. The patient subsequently required intubation for bronchoscopy and bronchoalveolar lavage (BAL). Cytology of the BAL revealed 56% eosinophils, consistent with eosinophilic pneumonia. The patient's respiratory distress stabilized on Solumedrol and the patient was extubated after one day. Her peripheral eosinophil percentage at this point was 22%. Along with the eosinophils identified on BAL and skin biopsy, her condition was consistent with hypereosinophilic syndrome. The patient gradually improved over the next few days on steroids and was eventually stable for discharge.

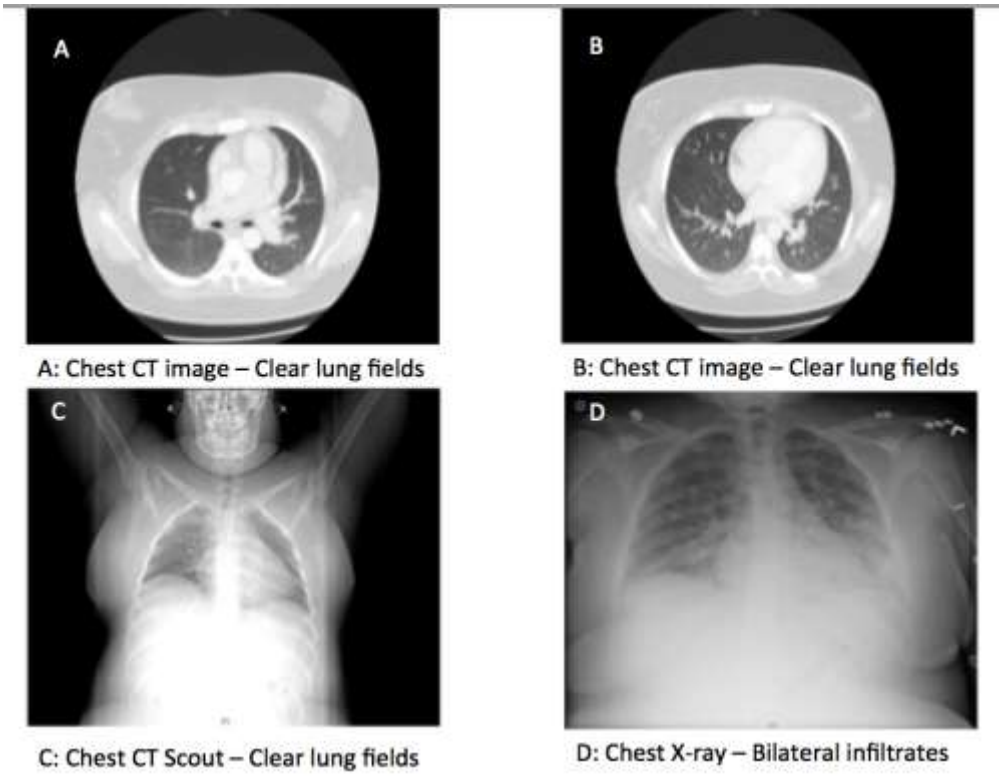

FIGURE 1.

DISCUSSION

Due to the sequence of events in this case and no other obvious sources, this could be the first reported case of doxycycline-provoked hypereosinophilic syndrome with no other identified precipitants leading to the development of significant eosinophilic pneumonia confirmed on BAL with 56% eosinophils. Historically, one of the side effects associated with minocycline has been systemic eosinophilia, but this association has not been made in the past with doxycycline.[1]

Eosinophilic pneumonias are generally characterized by pulmonary opacities without peripheral eosinophilia but increased eosinophilia on bronchoalveolar lavage or tissue biopsy. Eosinophilic pneumonias are classified between primary and secondary eosinophilic lung disease.[2] Primary eosinophilic pneumonias include simple pulmonary eosinophilia (aka Loeffler syndrome), acute eosinophilic pneumonia, chronic eosinophilic pneumonia, hypereosinophilic syndrome, and eosinophilic bronchitis.[2] Secondary eosinophilic pneumonias include allergic bronchopulmonary aspergillosis, bronchocentric granulomatosis, parasitic or fungal infection, reactions to drug or toxins, and eosinophilic vasculitis.[2] The pathology is unique to each presentation but generally there is eosinophilic infiltration of pulmonary tissue and increased inflammation of the parenchyma or airways. Symptoms can include shortness of breath, cough, fevers, chills, and malaise. Peripheral eosinophilia is not always present depending on the underlying disease process. In acute eosinophilic pneumonia, there is generally no peripheral eosinophilia as opposed to chronic eosinophilic pneumonia in which there is mild to severe peripheral eosinophilia and increased IgE serum levels. Bronchoalveolar lavage (BAL) is very important in diagnosis as > 25% eosinophils on BAL is diagnostic.[3] Differential diagnosis for eosinophilic pneumonia includes sarcoidosis, rheumatoid arthritis, pneumocystis jiroveci pneumonia, scleroderma, tuberculosis, lymphoma, interstitial pulmonary fibrosis, or acute respiratory distress syndrome if evaluated only by imaging and symptom presentation. The first line therapy for all presentations is high dose corticosteroids with taper. In disease specific treatment, IL-5 inhibitors like mepolizumab have been used, itraconazole in allergic bronchopulmonary aspergillosis to decrease steroid use, and anti-parasitics if there is an underlying parasitic infection.[2,4]

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