Case Report: Linear IgA Bullous Dermatosis Triggered by Amoxicillin-clavulanic acid

Bushra Salah Alraddadi1*, Tahani Magliah2, Yasser AlOtaibi3

1Senior dermatology resident, Ministry of Health, Madinah, Saudi Arabia
2Senior dermatology resident, King abdulaziz medical city, jeddah, Saudi Arabia
3Dermatologist, King fahad armed force hospital, Jeddah, Saudi Arabia

Abstract

Linear IgA bullous dermatosis (LABD) is rare autoimmune disease that can be caused with or without drugs. We present a 32 year old male with LABD secondary to Amoxicillin-clavulanic acid with complete recovery after the antibiotic was discontinued.

Keywords: Linear IgA Bullous disease, Vancomycin, Amoxicillin-clavulanic acid

Introduction:

Linear IgA bullous dermatosis (LABD) is a subepidermal autoimmune bullous disease characterized by linear IgA deposition at the basement membrane zone, which can be seen with direct immunofluorescence [1-4]. LABD classically presents with a widespread annular eruption of vesicles and tense bullae. Penicillin Antibiotics are usually prescribed to treat through infections and not from the most causative agents that trigger Linear IgA Bullous disease such as Vancomycin.

Case Report:

A 32 year old Saudi male was presented to the clinic with skin eruptions after five days of the Antibiotic use (Augmentin: Amoxicillin-clavulanic acid tablets 652 mg three times a day every 8 hours) due to an upper respiratory tract infection and fever. He started to develop skin rash at the axillary folds and then progressing to the trunk and face associated with severe itching and discomfort. No fever, systemic symptoms, abdominal pain or arthralgia were observed.

On examination, multiple tense bullae were scattered over the axillae, face, trunk and abdomen. These bullae were almost intact with serous fluid over normal skin (Figure 1). Negative Nikolsky sign and Asboe-Hansen sign were observed. Mucosa was free from any lesions. Nails showed normal examination. At the time of admission, he was vitally stable.

Laboratory tests were done and showed Leukocytosis with left shift neutrophilia, high Erythrocyte sedimentation rate, and otherwise normal hematology and chemistry results. Two biopsies were taken from the trunk. First was 3 mm punch biopsy for H&E in formalin which showed subepidermal cleft with dermal neutrophilic and some eosinophilic infiltrate (Figure 2). Second biopsy was for Direct immunofluorescence in gauze soaked in normal saline which showed linear deposition of IgA mainly and less IgG on the dermoeipidermal junction.

Copyright: © 2018 Bushra Salah Alraddadi, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.
Patient was advised to discontinue the Amoxicillin as it was the most likely the cause, we started him on Dapson 100mg once daily after a negative G6PD result. Patient reported clear of lesions after two days from starting therapy and was followed in our clinic weekly for blood tests and clinical assessment. On the next visit the bullous lesions were markedly improved showing only post inflammatory hyperpigmented patches, no new bullous lesions, and laboratory workup results were normal (Figure 3).

**Discussion and Conclusion:**

Linear IgA bullous dermatosis (LABD) is a subepidermal autoimmune bullous disease characterized by linear IgA deposition at the basement membrane zone, which can be seen with direct immunofluorescence [1-4]. LABD classically presents with a widespread annular eruption of vesicles and tense bullae.

Penicillin derivatives are usually prescribed and are not known to trigger LABD such as Vancomycin. We report this third case of linear IgA reported to be caused by Amoxicillin-clavulanic acid [5-6]. On the other hand, there is one other reported case of linear IgA caused by amoxicillin alone [7].

Linear IgA bullous dermatosis secondary to Amoxicillin-clavulanic acid is uncommon. A few cases of LABD-induced by Amoxicillin-clavulanic acid have been described in the literature.

**References:**