

Session/Poster#

Presenter

A53

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### **Vancomycin as a Likely Culprit of Linear IgA Bullous Dermatitis Among a Long List of Antibiotics**

#### Case description:

We report the case of a 59-year-old female diagnosed with linear IgA bullous dermatosis. She had initially been admitted for pyelonephritis and septic shock following mastoplasty 3 days prior. She was treated with empiric meropenem, piperacillin/tazobactam, and a full course of vancomycin for staphylococcus haemolyticus bacteremia. On day 3 (HD#3), she developed a new-onset, blistering rash over the right flank and right buttocks. Skin biopsy showed intraepidermal vesicular dermatitis with necrosis, not consistent with Stevens-Johnson Syndrome. Hydrostatic bullae were suspected. Subsequently, she developed multiple complications, and was treated with additional courses of piperacillin/tazobactam, vancomycin, meropenem, doxycycline, and ceftazidime/avibactam. A new-onset blistering rash over < 10% of the total body surface area was noted on HD#46. It was most prominent on the upper extremities and neck, its distribution resembling a "string of pearls". Direct immunofluorescence microscopy (DIF) indicated linear deposits of IgA along the epidermal basal lamina. Upon the cessation of vancomycin, her skin lesions healed spontaneously. She was labeled allergic to vancomycin and discharged home on HD#53.

Discussion: The incidence of LABD is estimated at approximately one per million per year. The pathogenesis of IgA-mediated inflammation in LABD entails the activation of neutrophils at the epidermal basal membrane. The suggested mechanism is two-pronged. First, it is a direct mechanism mediated via IgA immune complexes, which induce crosslinking of Fc $\gamma$ RI receptors, leading to direct activation and chemoattraction of neutrophils. Second, it is a complement-dependent mechanism, based on the C3 deposition, which can be visualized in approximately 30 % of cases. Since IgA lacks a C1q-binding site, it cannot activate the classical complement pathway. However, it can activate both the alternative and lectin pathways, which fuel the inflammation.