



## Poster Sessions

### P001

#### A CASE OF IPRATROPIUM BROMIDE ALLERGY IN A PATIENT WITH SEVERE PERSISTENT ASTHMA

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**Introduction:** Paradoxical bronchoconstriction with the use of inhaled anticholinergics is reported in the literature, and the mechanism is not fully understood. There are cases of patients allergic to soy and peanut having IgE-mediated reactions to ipratropium bromide, with hives and respiratory distress thought to be secondary to soy lecithin. We report a case of a 3-year-old Caucasian female who was admitted to National Jewish for severe-steroid dependent asthma. Her mother reported a history of an adverse reaction to ipratropium bromide. At that time, within 2 minutes of nebulized administration, her skin became diffusely erythematous, and she developed worsening respiratory distress. Her symptoms resolved within 30 minutes after treatment with diphenhydramine. She has no soy or peanut allergy and no history of urticaria.

**Methods:** She underwent puddle testing to ipratropium bromide with histamine and saline controls.

**Results:** Within 10 minutes, she developed diffuse hives and pruritus. She did not demonstrate respiratory distress. A dose of diphenhydramine resulted in full resolution of her symptoms.

**Conclusions:** Ipratropium bromide allergy is rare, and there is scarce literature in patients without peanut and soy allergy. Because of positive skin testing, this patient was advised to avoid all forms of ipratropium bromide, as this was thought to be a true IgE-mediated reaction, rather than a case of paradoxical bronchoconstriction. Further studies of allergy to ipratropium bromide in patients with asthma.

### P002

#### PREDICTIVE VALUE OF SKIN TESTING TO OMALIZUMAB AND EXCIPIENTS

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**Introduction:** The validity of allergy skin testing (AST) in predicting anaphylactic reactions to omalizumab has not been established. Our objective was to determine the positive predictive value (PPV) of AST in identifying subjects susceptible to anaphylaxis prior to starting omalizumab.

**Methods:** A retrospective study of subjects taking omalizumab over a six year period. Subjects were sorted into two cohorts; 1) AST prior to omalizumab to aqueous preparations of omalizumab, polysorbate 20, and commercially available mouse extract; 2) no pre-omalizumab testing. Intradermal and prick test results were combined as positive or negative to determine the PPV of AST.

**Results:** Incidence of anaphylaxis was 0.8%. 76% (n= 96 of 127) of subjects had negative AST and none developed anaphylaxis.

Of 31 subjects with a positive skin test, one subject (positive mouse AST) developed anaphylaxis 28 months after therapy initiation. In the non-AST cohort, one subject developed anaphylaxis 72 months after therapy initiation. Both reacted within 40 minutes and were treated successfully. Based on AST results, the PPV and specificity (CI 95%) were 3% (-15% to 21%) and 76% (69% to 83%).

**Conclusions:** We report that the longest time of onset of anaphylaxis after omalizumab initiation was six years. AST to omalizumab and major excipients is not warranted prior to starting therapy due to a low PPV. Since the risk of anaphylaxis remains low, 0.8%, and reactions frequently occur beyond the first three doses, the two hour wait period should be re-evaluated. Patients need to be monitored for 30 minutes and carry epinephrine auto-injectors.

### P003

#### A CASE OF FIXED DRUG ERUPTION RELATED TO NAPROXEN

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**Introduction:** Fixed drug eruptions (FDE) present as pruritic, well-circumscribed, erythematous macules that arise after exposure to specific medication. Lesions recur at the same site with each administration of the causative drug and evolve into hyperpigmented plaques. This delayed hypersensitivity reaction has been associated with multiple agents, primarily antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs). Typically, patients have multiple recurrences over an extended duration before diagnosis.

**Methods:** A retrospective chart review and prospective evaluation of a 40-year-old woman with an extended presentation of progressively worsening, intensely pruritic, maculopapular lesions was conducted. Dermatologic exam revealed multiple, large, hyperpigmented plaques, and scattered erythematous papules.

**Results:** A history of temporally associated, intermittent NSAID use, and punch biopsy demonstrating focal parakeratosis and superficial and deep perivascular infiltrate with eosinophils, led to a proposed diagnosis of FDE. Avoidance of all propionates, resulted in resolution of erythema and pruritus, and reduction of hyperpigmented plaques. Patch test to naproxen, triggered local inflammation of a former cutaneous lesion, but remained quiescent in previously unaffected skin.

**Conclusion:** The proposed mechanism of FDE is an intra-epidermal CD8+ T cell induced cytotoxic type IVc hypersensitivity reaction. IFN-gamma and cytotoxic granules damage keratinocytes, resulting in hyperpigmentation. Recruited Treg cells limit immune response and inhibit progression. Topical provocative testing is a safe alternative to oral challenge, as a means of identifying causative agents, although false negative results may occur. Maintaining a high level of suspicion for FDE, is the first step in eventual diagnosis.