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RESPIRATORY

READER

THE DIVISION OF PULMONARY, ALLERGY AND CRITICAL CARE MEDICINE AT UPMC

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Dear Colleague,

The UPMC Comprehensive Allergy-Immunology Clinic and University of Pittsburgh Asthma Institute at UPMC are premier clinical and research programs dedicated to the advanced and comprehensive care of patients suffering from these conditions. The clinical teams consist of pulmonary and allergy/immunology specialists who treat a wide spectrum of conditions. Our pulmonary physicians focus on the evaluation and treatment of complex airway disease with an emphasis on immunologic manifestations of airway disease and severe asthma. Our allergy/immunology physicians evaluate and treat allergic and immunologic conditions that can affect one or multiple organ systems. They specialize in the evaluation and treatment of the following conditions:

- Severe allergic upper and lower airway disease
- Refractory chronic urticaria
- Histaminergic and bradykinin-mediated angioedema
- Multiple drug allergies
- Systemic eosinophilic disorders
- Idiopathic allergic reactions
- Primary and secondary immunodeficiency conditions

In this issue of the *Respiratory Reader,* you will read about unique allergy and upper airway procedures that are offered at our center. Additionally, you will learn about the cutting-edge translational and clinical asthma research performed by our world-renowned researchers. Finally, our fellow has a fascinating case that was recently seen by our allergy/immunology service. We hope that you will enjoy this issue.

With great enthusiasm and respect,

Alison Morris, MD, MS
Chief, Division of Pulmonary, Allergy and Critical Care Medicine
Professor of Medicine, Immunology, and Clinical & Translational Research
Director, Center for Medicine and the Microbiome
Vice Chair for Clinical Research, Department of Medicine
UPMC Endowed Chair in Translational Pulmonary and Critical Care Medicine

Andrej A. Petrov, MD
Associate Professor of Medicine
Section Chief of Allergy
Division of Pulmonary, Allergy and Critical Care Medicine

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UPMC LIFE CHANGING MEDICINE

Case Presentation: Successful Treatment of Steroid-Refractory Hypereosinophilia With Reslizumab

by Kara E. Coffey, MD

Hypereosinophilia (HE), defined by a persistent absolute eosinophil count (AEC) of greater than 1,500 cells/uL, has a broad differential with primary or secondary etiologies.[1] Hypereosinophilic Syndrome (HES) is associated with end-organ damage, and first-line treatment is corticosteroids, though the response may be variable.[2] Monoclonal antibodies against IL-5, important for eosinophilic development,[3] present another therapeutic target.[4,5] This case describes a patient with profound steroid-refractory HE who was successfully treated with reslizumab.

The patient is a 72-year-old male Vietnam veteran with history of relapsing polychondritis, myasthenia gravis (MG) on chronic prednisone, and remote seizure disorder. He presented to the emergency department with eight weeks of progressive weakness, fatigue, and 10 kg weight loss, with new and worsening dyspnea in the last week. He had an elevated white blood cell (WBC) count of 25.6 x 10^9/L with an AEC of 2.3 x 10^9/L (9% of differential) (Figure 1a). A chest radiograph showed multifocal opacities and, within hours, he developed respiratory failure requiring intubation and mechanical ventilation (Figure 1b). A chest CT scan demonstrated patchy bilateral consolidations and ground-glass opacities (Figure 1c).

The patient was treated with methylprednisolone 125 mg IV once and subsequently maintained on prednisone 80 mg/day. He received multiple antibiotics for presumed infectious pneumonia, though cultures and cytology from bronchoscopy were negative; however, cell counts revealed 22% eosinophils. WBC trends showed rising eosinophilia, peaking at AEC of 12.68 x 10^9/L (40% of WBC) despite high-dose prednisone.

Tryptase, troponin, antineutrophil cytoplasmic antibodies (ANCA), rheumatoid factor (RF), antinuclear antibodies (ANA), Strongyloides titers, and anti-cyclic citrullinated peptide (anti-CCP) studies were negative. Preliminary pathology of bone marrow biopsy showed mild hypercellularity with no increased blasts; peripheral blood flow cytometry showed no CD34+ myeloblasts.

Despite treatment with high-dose prednisone and discontinuation of multiple medications, the patient's HE persisted (Figure 1a). After two weeks of high-dose corticosteroids, he received anti-IL5 treatment with reslizumab, dosed at 3 mg/kg. His AEC prior to reslizumab infusion was 8.73 x 10^9/L (43%); the next day, his AEC was 1,000 cells/uL (6%), and he was transferred out of the intensive care unit (Figure 1a). His WBC showed no detectable eosinophils six days post-reslizumab.

Final cytogenetic studies showed no clonal abnormalities with negative FIP1L1-PDGFRA, JAK2, and BCR-ABL testing. Next-generation targeted sequencing detected missense and frameshift mutations in SRSF2 and TET2, respectively. These results supported the diagnosis of chronic eosinophilic leukemia, not otherwise specified (CEL-NOS), as TET2 mutations have previously been associated with CEL-NOS.[6] However, idiopathic HES

(I-HES) was an alternative diagnosis given the lack of increased blasts or chromosomal abnormality. This case presents a dilemma in the classification of primary HE, as TET2 mutations have been seen in CEL-NOS.[6] However, the lack of blasts in the periphery and bone marrow would argue against an underlying neoplasm. Although inferior to I-HES without mutation, patients with I-HES with mutation have a similar survival rate to patients with CEL-NOS.[6] Nine months post-reslizumab, the patient's AEC remains suppressed and the prednisone was tapered to 10 mg/day for MG treatment (Figure 1a).

Reslizumab was selected for its IV delivery and weight-based dosing. Mepolizumab has been used intravenously at higher doses for HES in clinical trials, but commercially is available for subcutaneous administration.[5] In a small study of four patients with HES who were treated with reslizumab dosed at 1 mg/kg, two patients had an initial favorable clinical response, though the AEC and symptoms rebounded within eight weeks.[7] Additionally, a case report of one patient with eosinophilic dermatitis received monthly reslizumab 3 mg/kg with significant improvement.[8] In this patient, the symptom improvement and eosinophil suppression has lasted over nine months without relapse after a single reslizumab infusion at 3 mg/kg. Additional studies are required to explore the efficacy of higher-dose reslizumab for treatment of hypereosinophilic disorders.

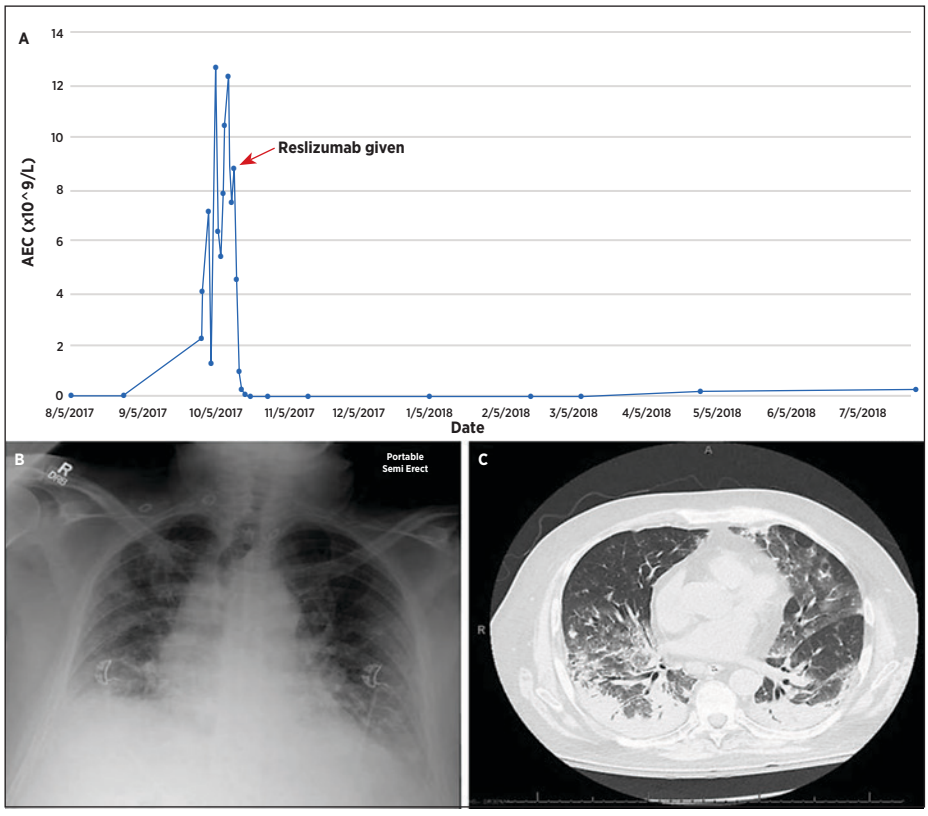

Figure 1: (A) Absolute eosinophil count (AEC) over time. **(B)** Chest X-ray showing multifocal opacities. **(C)** Chest CT scan showing patchy bilateral consolidations, ground-glass opacities and scattered nodules.

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Approach to the Patient with Drug Allergy

by Laura J. West, MD

Adverse drug reactions (ADRs) are defined by the World Health Organization as "any noxious, unintended, and undesired effect of a drug that occurs at doses used for the prevention, diagnosis, and treatment." The incidence of ADRs is not uncommon, with studies reporting an incidence of 2-7% in hospitalized patients.[1-2] The monetary costs to society due to ADRs have been estimated to range from $75–$180 billion each year for adults.[3]

Types of Drug Allergies

ADRs can be categorized in two different groups. Type A reactions include common and predictable reactions (e.g., renal toxicity, abdominal pain, etc.) and occur in normal individuals. Type B reactions, also known as drug hypersensitivity reactions, are defined as objectively reproducible signs or symptoms initiated by a drug at a dose usually tolerated by normal subjects.[4]

Drug allergy is mediated by immunologic mechanisms. IgE-mediated, or immediate drug reactions, usually develop within a couple of hours of drug exposure and manifest with allergic symptoms including urticaria, angioedema, wheezing, and gastrointestinal symptoms that can progress to anaphylaxis. These reactions are limited in duration and usually last for up to 24 hours. Delayed-type drug hypersensitivity is a T-cell-mediated process and manifests as a cutaneous eruption that develops more than 24 hours after drug exposure. Unlike IgE-mediated drug reactions, these usually last for days to weeks.

Non-allergic drug hypersensitivity includes reactions provoked by non-immunologic mechanisms such as angiotensin-converting enzyme (ACE) inhibitor-induced angio-edema, or COX-1 inhibition in aspirin-exacerbated respiratory disease (AERD).

The differential for non-immediate drug reactions is broad and includes fixed drug eruptions, lichenoid drug reactions, drug-induced vasculitis, drug reaction with eosinophilia and systemic symptoms (DRESS), serum sickness, bullous pemphigoid, pemphigus vulgaris, Stevens-Johnson syndrome, and toxic epidermal necrolysis.

Approach to Drug Allergy Evaluation and Testing

A thorough history is imperative in the evaluation of the drug-allergic patient. Key components of the history include the type of medication, temporal relationship between drug exposure and onset of symptoms, history of prior drug exposure, propensity of a given drug to cause a reaction, and characterization of reaction type. A complete physical examination is also essential; maculo-papular eruptions are more characteristic of non-immediate reactions whereas urticarial lesions are the predominant skin eruption in immediate reactions.

Drug Challenges and Desensitization

Drug challenges and desensitizations can be performed by an allergist/immunologist. Skin testing in the work-up of an ADR is indicated in patients with symptoms suggestive of an immediate drug allergy (e.g., flushing, urticaria, angioedema, anaphylaxis, conjunctivitis, rhinitis, or bronchospasm). For example, this procedure is frequently performed when evaluating a patient with a history of hives after taking penicillin or cephalosporin antibiotics. If skin prick and intradermal testing are negative, the next step in evaluation is administration of a graded oral medication challenge under close observation.

In certain circumstances, patients may require treatment with a drug despite a documented immediate drug hyper-sensitivity. Drug desensitization is defined as a procedure that induces temporary tolerance to a drug, allowing a patient to receive the optimal agent and dose for treatment of their disease.[5] During drug desensitization, tolerance is achieved by delivering incremental doses at fixed intervals until the final drug dose is achieved. Of note, drug desensitization does not induce long-term drug tolerance, and patients must remain on daily administration of the drug to maintain tolerance. Once the drug is discontinued, desensitization is again required upon all subsequent drug exposures.

Management of Drug Allergies at UPMC

The UPMC Comprehensive Allergy-Immunology Clinic is uniquely equipped to evaluate and manage patients with adverse drug reactions. Allergy physicians will often perform medication skin testing to

determine the culprit medication, for example in patients who had perioperative anaphylaxis after receiving multiple medications at the same time. Additionally, the inpatient drug desensitization program exists at multiple hospitals where desensitizations are performed in critically ill patients. These drug desensitizations can include antibiotics, monoclonal antibodies, chemotherapeutic agents, and cardiac medications. UPMC allergy physicians have also implemented an outpatient drug desensitization program, thereby removing the need for hospital admission. Besides antibiotic and other outpatient medication desensitizations, a three-day protocol with serial pulmonary function tests is also performed in patients with AERD who have severe nasal polyposis. It is noteworthy that many of these desensitization protocols are novel and have been created specifically to address complex patient care scenarios.

Some of these protocols include rituximab desensitization for mucosa-associated lymphoid tissue (MALT) lymphoma in the setting of serum sickness,[6] omalizumab desensitization in patients with severe asthma,[7] aspirin and clopidogrel desensitizations in cardiac patients,[8,9] ferric carboxymaltose desensitization in patients with refractory iron deficiency anemia,[10] and novel pre-treatment protocol in patients with IV dye allergy, refractory to the standard pre-medication.[11]

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Vocal Cord Dysfunction

by Andrej A. Petrov, MD

Vocal cord dysfunction (VCD) is a functional disorder of the vocal cords that causes respiratory and laryngeal symptoms.[1] Laryngeal hyperresponsiveness, induced by the combination of psychologic, environmental, and comorbid factors, leads to episodic and exaggerated laryngeal constriction.

The most common presenting symptoms of VCD are dyspnea, cough, chest tightness, and wheezing (Table 1).[2] VCD presentation can overlap with muscle tension dysphonia, chronic cough, and globus pharyngeus conditions.[2] VCD has also been recognized as an asthma mimicker, leading to misdiagnosis of asthma for an average of nine years.[3] Additionally, VCD can coexist with asthma in one-third of cases. Importantly, VCD patients misdiagnosed with asthma had higher health care utilization rates — including emergency department visits, oral steroid and inhaler use, and chest CT imaging studies — compared with patients with VCD who did not mimic asthma.[4] Finally, VCD can also mimic allergic reactions — such as anaphylaxis, drug allergies, and laryngeal angioedema — as patients with these reactions frequently report throat tightness as their main symptom.

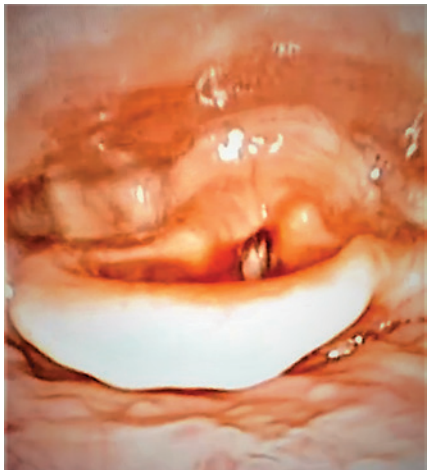

Figure 1: Laryngoscopy, the gold standard for diagnosis of VCD, allows for the direct visualization of adduction of vocal cords during inspiration.

The gold standard for VCD diagnosis is laryngoscopy that shows adduction of vocal cords during inspiration (Figure 1).[2] However, laryngoscopy can frequently be normal if the patient is asymptomatic. Therefore, to make the correct diagnosis, laryngoscopy with provocative maneuvers to trigger VCD symptoms is recommended. Additionally, UPMC allergy researchers have developed the Pittsburgh Vocal Cord Dysfunction Index, a four-question scoring system, to help distinguish VCD from asthma. The index has 83% sensitivity and 95% specificity in distinguishing vocal cord dysfunction from asthma (Figure 2).[2]

The subtypes of VCD include spontaneous VCD, irritant VCD, somatic VCD, and exercise-induced laryngeal obstruction (EILO).[2] EILO is a unique subtype of VCD where symptoms occur only with physical activity, and resting laryngoscopy is usually normal. It has been studied mostly in adolescents and young, athletic adults.

Table 1: Vocal Cord Dysfunction Symptom Profile

Symptom	Patients (%)
Dyspnea	75-98
Cough	40-80
Wheezing	50-80
Chest Tightness	15-75
Chest Pain	12-22
Throat Tightness	15-65
Dysphonia	25-60
Stridor	10-50
Difficulty Swallowing	30

Common presenting symptoms of VCD.

The prevalence in older adults is unknown at this time. The pathophysiology of EILO is explained by the collapse of the laryngeal cartilage when exposed to high-volume airflow during vigorous exercise, resulting in breathing problems. The most common symptom is exertional dyspnea, seen in more than 95% of patients, followed by stridor and/or hoarseness in 54% of patients.[5] The symptoms in EILO typically occur at the peak of physical activity and can be very short-lived. Therefore, post-exercise laryngoscopy may fail to detect the upper airway obstruction. Continuous laryngoscopy is a procedure during which laryngoscopy is performed during exercise, and the motion of glottis and supraglottis is observed in real time (Figure 3).

Pittsburgh VCD Index

Throat Tightness	4
Sensitivity to Odors	3
Dysphonia	2
Absence of Wheezing	2

Figure 2. Pittsburgh VCD Index: Score of a ≥ 4 during respiratory symptoms indicates VCD.

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Figure 3: Patient undergoing a continuous laryngoscopy while exercising.

Upper Airway Exercise Breathing Center at UPMC

The **Upper Airway Exercise Breathing Center at UPMC**, which launched in January 2020, offers Continuous Laryngoscopy Evaluation (CLE) during exercise for amateur and elite athletes, as well as nonathletes, with unexplained exertional shortness of breath. Patients 14 years and older are evaluated with state-of-the-art diagnostics and receive treatment specifically designed for people with breathing problems while exercising. UPMC is the second academic medical center in the United States to offer this diagnostic procedure for exercise-induced laryngeal obstruction (EILO). Other tests include exercise spirometry, methacholine challenge test (with laryngoscopy), and cardiopulmonary exercise testing with CLE. Additionally, therapeutic respiratory retraining techniques via biofeedback will be provided during CLE.

Diagnostic CLE tests at UPMC are performed by a multidisciplinary Allergy/Respiratory/ENT team. Our experts include Andrej A. Petrov, MD, from the Division of Pulmonary, Allergy and Critical Care Medicine, and Jackie Gartner-Schmidt, PhD, CCC-SLP, a dyspnea-specialized speech-language pathologist and director of the Speech-Language Pathology-Voice Division in the Department of Otolaryngology and co-director of the UPMC Voice Center.

Exercise is important. Whether your patients are working out for general health benefits or competition, our center helps patients achieve their goals.

Referrals to the Upper Airway Exercise Breathing Center at UPMC can be made to **ExerciseBreathing@upmc.edu**, or call **412-692-4557**.

Treatment Options for Allergic Rhinitis and Conjunctivitis

by Merritt L. Fajt, MD, and Andrej A. Petrov, MD

Allergic rhinitis and conjunctivitis (ARC) are one of the most common medical conditions that affect both children and adults. While ARC does not cause significant morbidity or mortality, its impact on quality of life cannot be overstated. The symptoms of ARC interfere with cognitive and sleep function, cause fatigue and irritability, and are a common cause of missed days of school and work.

Many patients will report feeling like they have a never-ending common cold. Therefore, ARC is also one of the most common reasons for physician office visits. Additionally, allergic rhinitis and post-nasal drip are frequently associated with chronic cough, and uncontrolled allergic rhinitis can worsen asthma symptoms.[1]

ARC Medications and Immunotherapies

Allergen avoidance represents a simple and inexpensive way to treat allergic symptoms. Nonetheless, modifying allergen levels in the environment can be challenging, especially when it comes to decreasing exposure to pollens and dust mites. Another common cause of allergic symptoms is animal or pet allergy. Because patients are frequently emotionally attached to their pets, their removal is often not an option. ARC can also be treated with over-the-counter (OTC) or prescription medications. These include oral, nasal, and eye decongestants and antihistamines, intranasal steroid sprays, leukotriene modifiers, and nasal irrigation with saline.

For patients who fail medical therapy or who wish to decrease their medication use, allergen immunotherapy (AIT) is recommended. AIT can be administered sublingually and subcutaneously. Sublingual allergen immunotherapy (SLIT) tablets are available only for select allergens: dust mites, grass, and ragweed pollens. These allergens cannot be combined and therefore SLIT is used mostly in patients who have only one allergic trigger. Subcutaneous allergen immunotherapy (SCIT) or allergy shots can be administered in the allergy or general practice office. The patients can be desensitized to many allergens at once and the benefits include decrease in symptoms and medication use as well as induction of tolerance.[2]

Novel SCIT Protocol Developed at UPMC

SCIT administration is divided in build-up and maintenance phases. During the build-up phase, patients receive weekly injections while during the maintenance phase, they receive monthly injections. Prior data on SCIT has shown difficulty with patient compliance due to the length of the build-up phase (four to eight months) and a delay in symptom relief. At the UPMC Comprehensive Allergy-Immunology Clinic, we have developed a novel accelerated SCIT protocol that decreases the build-up time to maintenance dose by 50%, resulting in more rapid and safe relief of allergic symptoms. It consists of one-day, eight-hour protocol followed by shortened build-up to the full maintenance dose. Patients are treated the day before and the day of the rapid protocol with prednisone 40 mg, montelukast 10 mg, and non-sedating H1 and H2 antihistamines. As a safety precaution, all patients are prescribed auto-injectable epinephrine.

(Continued on Page 10)

Lab Spotlight: Bedside to Bench and Back Again

by Sally Wenzel, MD, and Anuradha Ray, PhD

The last three years have produced four new "Type-2" biologic agents for the treatment of severe asthma. Overall, these drugs are substantially improving the lives of patients with this disease, including patients who previously required systemic corticosteroids on a daily basis.

These four drugs interfere with pathobiologic/immune "Type-2" pathways, which contribute to eosinophilic airway inflammation mucus production and secretion. Their development arose through the combination of basic bench-driven research, improved clinical-translational understanding of asthma as a heterogeneous disease, and, importantly, clinical trials. None of this could have happened without an integration of efforts.

Most studies now suggest that Type-2 (T2) inflammation (involving the cytokines IL-4, IL-5, and IL-13) drives at least 50% of severe asthma patients. Patients with this inflammation type display elevations in T2 biomarkers like blood eosinophils and fractional exhaled nitric oxide (FeNO). However, even in patients with these biomarkers, responses to these drugs can be variable or even non-existent. To effectively treat a chronic disease like asthma, less expensive alternatives are needed.

Much of the basic research on severe asthma at the University of Pittsburgh has focused on these clinical observations and studies that show severe asthma is heterogeneous and is characteristically poorly controlled by corticosteroids. Therefore, a one-size-fits-all approach is inadequate to control disease symptoms in all asthmatics. Given this understanding, we are using cutting-edge tools in immunology and molecular biology to understand the cellular and molecular underpinnings of disease heterogeneity in humans. We are also leveraging the knowledge gained from these studies to develop pre-clinical models in mice to study the importance of some of these pathways in vivo as targets for future therapy. As an example, our human-based studies have identified the presence of a heightened Type 1/IFN-γ response in the airways of a subset of severe asthma patients, many of whom also show elevated expression of T2 cytokines in their immune cells.

This knowledge was used to develop a mouse model of severe asthma that recapitulates both immunological features of the disease in these patients as well as the poor response to corticosteroids. The animal model has helped us to identify a potential role for IFN-γ in poor lung function in disease and an IFN-γ-triggered mechanism that may partly contribute to unresponsiveness to corticosteroids in these patients. It appears that cooperation between the glucocorticoid receptor (GR), a nuclear hormone receptor, to which corticosteroids bind to exert their effects, and the IFN-γ-activated transcription factor STAT1 in target cells, such as airway epithelial cells and monocytes and macrophages, promote the expression of pro-inflammatory genes such as CXCL10, which, along with T2 cytokines, may perpetuate airway inflammation in these patients. Clearly, these asthmatics need alternative therapies to block the unwarranted immune activation in their airways. New insights gained into molecular interactions at play in their immune and epithelial cells will help guide the development of future therapies.

Collectively, these studies highlight the importance of using clinical observations to inform basic research, the results of which will serve as building blocks for the future of precision medicine in asthma. Our ultimate goal is to provide the best treatment for each endotype of asthma.

To refer a patient or request a patient transfer to the University of Pittsburgh Asthma Institute at UPMC, call **412-648-6161**.

For more information on our department's involvement with asthma clinical trials, please call **412-647-9955** or **1-866-804-5278**, or visit our website at **http://asthmainstitute.pitt.edu.**

Meet the Clinical Team

Allergy/Immunology:
Andrej A. Petrov, MD
Merritt L. Fajt, MD
Theresa Nee, RN
Erin Corrigan, RN

Pulmonology/Asthma:
Sally Wenzel, MD
Marc Gauthier, MD
Cathy Vitari, RN

Pulmonology/Asthma: Cathy Vitari, RN; Sally Wenzel, MD; Marc Gauthier, MD

Update on Asthma Clinical Trials

by Sally Wenzel, MD

The University of Pittsburgh Asthma Institute at UPMC is one of the most active centers for clinical studies in asthma in the country. National Institutes of Health (NIH) studies are focused on the natural history of severe asthma and biologic factors that alter its course, as well as improving understanding of asthma heterogeneity. Results from the longitudinal Severe Asthma Research Program found that mucus plugging, which can reliably be identified on lung CT imaging, is a critical feature of many severe asthma patients and is present even in the absence of cough and sputum.[1] This mucus pattern on CT imaging is associated with more Type-2 inflammation and higher sputum eosinophils. Further studies will determine whether Type-2 targeted biologic agents improve this mucus plugging and clinical outcomes. Ongoing industry trials are focused on determining whether moving upstream of IL-4, IL-5, and IL-13 might lead to even better outcomes, with studies of antibodies directed to the IL-33 and thymic stromal lymphopoieten (TSLP) pathways. TSLP antibodies have already been reported to improve asthma,[2] but are not yet approved. Additionally, small molecule approaches developed at the University of Pittsburgh are also being studied. If efficacious, these drugs would be less expensive than antibody approaches. Finally, asthma is associated with poor sleep and nocturnal awakenings, although it is unclear whether the asthma causes the awakenings or poor sleep worsens the asthma. Faith S. Luyster, PhD, assistant professor at the University of Pittsburgh's School of Nursing, is performing an NIH study of cognitive behavioral therapy to improve sleep in poorly controlled asthma. Her studies will determine whether improving sleep improves asthma control. For more information on our department's involvement with asthma clinical trials, please call **412-647-9955** or **1-866-804-5278**, or visit our website at **http://asthmainstitute.pitt.edu**.

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Treatment Options for Allergic Rhinitis and Conjunctivitis *(Continued from Page 8)*

In our recently published study, "A 10-year experience of a novel and safe modified environmental rush immunotherapy protocol (MERIT)," we presented our experience with this protocol.[3] The study was a retrospective analysis of 362 adult patients with allergic rhinitis seen over a 10-year period (January 2005 to January 2015) who underwent the MERIT protocol. Our population consisted of 230 females and 132 males with a median age of 33 years and body mass index (BMI) of 26.2. Approximately 48% (n=173) of patients had a diagnosis of asthma. A history of urticaria and angioedema was seen in 39 (10.8%) and 5 (1.4%) patients, respectively. In total, 50 patients (13.8%) experienced systemic reactions at any time during AIT, with 4.7% occurring on the MERIT day. Most of the systemic reactions were mild and were rapidly reversed with treatment. While there was no association with gender, the group who experienced a systemic reaction tended to be younger in age and had a lower BMI. There was also a tendency for an association between systemic reactions and a diagnosis of urticaria or angioedema. Asthma has traditionally been identified as a risk factor for systemic reactions; however, our study found no association with asthma and systemic reactions to AIT. The occurrence of a systemic reaction did not impact premature discontinuation of AIT. In terms of AIT content, 71% had both seasonal and perennial allergens in their extract. Dust mites were the most commonly included allergen (in 75%), followed by cat (66%), any weed pollen (65%), and grass mix pollen (61%). In multivariate analysis of allergen content by systemic reactions, significant associations were seen with extract containing dust mites, cat, and mugwort (a weed pollen). Our unpublished data also show that there is no difference in risk between traditional and MERIT protocols.

In summary, ARC is one of the most common medical conditions with a significant impact on quality of life. The treatment options include allergen avoidance, medical therapy, and allergen immunotherapy. At UPMC, we offer a novel modified rush allergen immunotherapy protocol that allows rapid and safe outpatient relief of refractory allergic symptoms.

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Allergy-Immunology Research Studies

by Merritt L. Fajt, MD, and Andrej A. Petrov, MD

The most common cause of chronic idiopathic urticaria and angioedema is autoimmune where mast cells are activated by autoimmune antibodies targeting mast cells. At the UPMC Comprehensive Allergy-Immunology Clinic, there is an ongoing research study evaluating the efficacy of omalizumab (anti-IgE monoclonal antibody), anti-inflammatory (colchicine, sulfasalazine, hydroxychloroquine, and sulfasalazine) and immunosuppressive (mycophenolate) treatments on refractory urticaria.

Secondary hypogammaglobulinemia is a common complication of immuno-suppressive therapy. In collaboration with the UPMC lung transplant pulmonologists, there is an ongoing study evaluating the effects of end-stage lung disease and immunosuppressive medications on immunoglobulin levels and possible clinical complications in patients before and after lung transplantation.

Allergy-Immunology: Andrej A. Petrov, MD; Merritt L. Fajt, MD; Theresa Nee, RN; Erin Corrigan, RN

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Conference Directors:
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Please visit the conference website often for updates, as they become available: **https://lungconf.pitt.edu**. It will offer many other details regarding the conference, travel, and our Pittsburgh area in general. A call for abstracts portal will also open on this site in Spring 2020. We hope you will consider submitting an abstract and joining us as a registrant in October!

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The Division of Pulmonary, Allergy and Critical Care Medicine at UPMC

Comprehensive Lung Center
3601 Fifth Ave., Fourth Floor
Pittsburgh, PA 15213
T: 412-648-6161
F: 412-648-6869

Alison Morris, MD, MS
Chief, Division of PACCM

EDITORS
Christopher Faber, MD
Theresa Dobransky

ADDRESS CORRESPONDENCE TO:
Theresa Dobransky
Division of PACCM
3459 Fifth Ave.
Pittsburgh, PA 15213

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