

## ABSTRACTS: CONCURRENT SESSIONS

### 29 Effects of cyclosporin in the treatment of Lupus Pulmonary Hypertension D.J. Yoshii, DO,\*J.J. Joyce, MD, M. Keller, MD, C.K. Oh, MD Harbor-UCLA, Torrance, CA

Pulmonary hypertension (PHTN) is an uncommon complication of systemic lupus erythematosus typically occurring after years of active disease. Chronic Mucocutaneous Candidiasis (CMCC) is on the other hand a disease associated with recurrent cutaneous Candida infections. The two diseases have not previously been reported together.

We are reporting a 13-year-old Hispanic female who had previously been diagnosed with CMCC who developed Lupus PHTN. The patient first presented at 11 mo of age with an extensive Candidal rash of the axilla. She initially was treated as an inpatient but was lost to follow up. During this time the child had persistent onychomycosis and thrush but was otherwise healthy. At 12 y/o the patient developed hepatomegaly, malar rash, pericardial effusion, and difficulty breathing. She was hospitalized for evaluation and treatment. The ANA was strongly positive, and the patient was found to have suprasystolic PHTN. Lupus anticoagulants were negative.

The patient responded to methylprednisolone and the PHTN decreased to 50% of systemic pressures. When the patient was changed to oral prednisone her pulmonary pressures would increase to 80% systemic pressures within 1 week. She was admitted for more IV methylprednisolone. Steroids quickly became ineffective and pulse cyclophosphamide (CYT) was started. Pulse CYT did little to change the PHTN. Oral cyclosporin (CYA) was started at with daily CYT. The patient responded well to the new therapy, pulmonary pressures decreased to 50% of systemic blood pressure. However the patient then developed ARDS from a severe fungal and CMV pneumonia and died. In summary, our data suggests CYA but not CYT effectively suppressed PHTN in steroid resistant Lupus PHTN.

### 30 ERADICATION OF *CHLAMYDIA PNEUMONIAE* FROM BRONCHOALVEOLAR LAVAGE (BAL) FLUID ASSOCIATED WITH ASTHMA IMPROVEMENT: CASE REPORT. D.L. Hahn, MD\*; K.L. Middleton, MD, Madison, Wisconsin; L.A. Campbell, PhD; S-P. Wang, MD, Seattle, Washington

**Background:** *Chlamydia pneumoniae* (*Cpn*) has been associated with asthma by serology and organism identification in upper respiratory secretions. **Objective:** Correlate *Cpn* PCR findings in upper and lower respiratory secretions with clinical status before and after antimicrobial therapy of adult-onset asthma. **Methods:** Nasopharyngeal swab (NP) and BAL fluid were obtained from a nonatopic, nonsmoking 65 year old male with stable moderate persistent adult-onset asthma before (week 0) and after (week 12) azithromycin therapy (1000 milligrams orally, once per week during weeks 1-5). NP and BAL were tested for *Cpn* PCR; BAL was also cultured for *Mycoplasma pneumoniae*, respiratory viruses and pyogens. Baseline asthma status (FEV1, PEFR, symptoms, Juniper Asthma QOL and medication use) was compared to 8 months after treatment. **Results:** Baseline chest and sinus xrays were normal. Pre-treatment BAL was positive for *Cpn* PCR; all other pre-treatment and all post-treatment specimens were negative for pathogens. *Cpn* IgG antibody titers remained 1:512. Pre-bronchodilator FEV1 increased (2.77 L to 3.14 L); AM and PM PEFR increased (449 to 497 L/min and 484 to 537 L/min, respectively). Symptoms improved and Juniper Asthma QOL score improved from 3.97 to 4.94 (7 point scale). Medication use decreased (9 puffs to 6 puffs of ICS, inhaled ipratropium discontinued). **Conclusions:** This is the first report of asthma improvement following microbiologic eradication of *Cpn* from BAL fluid. Results of sampling the upper respiratory tract may not correlate with presence of *Cpn* in the lung.

### 31 URTICARIA AND THYROID CARCINOMA: A NEW ASSOCIATION? A. Segalene, MD\* and A. Gewurz, MD, Rush-Medical Center and Cook County Hospital, Chicago IL

Although the pathogenesis of chronic urticaria may be linked to underlying infection or autoimmune disease, particularly thyroiditis, several studies have failed to show an association with carcinoma. We report a 59-year-old Byelorussian woman who presented 1 yr ago with intermittent urticaria and dysphagia of 3 months' duration. Her only current medication was cetirizine 10 mg QD PRN. A "thyroid disease" had been diagnosed in Russia 20 years earlier, but no treatment was given. The past medical history was otherwise unremarkable. The family history was negative for urticaria or endocrinopathy. The social history was noncontributory, and review of all other systems was negative. Physical examination, including the thyroid gland, was completely normal, except for several small urticaria. Laboratory tests showed normal CBC, ESR, serum electrolytes and urinalysis. Testing for thyroid disease showed thyroid stimulating hormone 33.6  $\mu$ IU/ml (normal, 0.4-4.2); T4 1.24  $\mu$ g/dl (normal, 5.8-11.0), antimicrobial antibody index 1.19 (positive >0.38) and negative antithyroglobulin antibody. Thyroid ultrasonography demonstrated diffuse nodular hyperplasia, and a biopsy revealed chronic lymphocytic thyroiditis and localized papillary adenocarcinoma (follicular variant). The patient was treated with partial thyroidectomy and thyroxine replacement. Within a week both the urticaria and dysphagia disappeared and have not recurred. It was concluded that she had urticaria associated with thyroid adenocarcinoma, autoimmune (Hashimoto's) thyroiditis and secondary hypothyroidism.

As shown by this case, the clinical associations of chronic urticaria may include carcinoma, as well as autoimmune disease, of the thyroid. In a patient with hypothyroidism and thyroid autoantibodies, a coexisting thyroid malignancy could be overlooked without an ultrasonogram or other diagnostic test.

### 32 PANDAS OR PARC? A PERPLEXING PEDIATRIC PROBLEM. M. Devera, MD\* and A. Gewurz, MD, Rush Medical Center-Cook County Hospital A/I Program, Chicago IL

An 11YO boy presented with frequent, spasmodic blinking, squinting, mouth stretching and occasional upward rolling and sideways twisting of the neck of 6 months duration. He was examined by a neurologist, who observed "multiple motor tics." There was no other indication of neuromuscular or psychiatric abnormality. The patient had high levels of serum IgG antibodies against streptococci and was given an initial diagnosis of pediatric autoimmune neuropsychiatric disease associated with streptococci or PANDAS (*Am J Psychiatry* 1997;154:110-2). Daily oral penicillin prophylaxis against streptococcal reinfection was initiated. The patient also complained of mild itching of the eyes and nose and postnasal drip with throat-clearing, which were partially relieved by diphenhydramine; he denied associated sneezing, nasal/sinus congestion, rhinorrhea or tearing. The past medical and family history were negative for atopic (rhinosinusitis, conjunctivitis, asthma or eczema) or rheumatologic diseases. The patient had no known hypersensitivity to foods, drugs or other antigens. He formerly had a pet cat. Physical examination showed a healthy-looking child with infrequent facial tics (staccato blinking, followed by a "startled" expression), allergic shiners, edema of the nasal mucosa and mild lichenification of antecubital skin. Slit lamp examination by an ophthalmologist revealed conjunctivitis with mild tarsal papillary reaction. Skin testing was positive for immediate reactivity to cat and dustmite allergens. Our diagnosis was blepharospasm secondary to perennial allergic rhinitis and conjunctivitis (PARC) masquerading as PANDAS. All symptoms improved following treatment with daily cetirizine, montelukast, intranasal mometasone and olopatadine eyedrops; environmental control, and twice-weekly dustmite allergen vaccine immunotherapy. We recommended discontinuation of oral penicillin prophylaxis.