several risk factors that would increase her risk of angioedema while on ACE inhibitors (and potentially DPP-IV inhibitors). Generally, risk factors associated with ACE inhibitor-induced angioedema include previous or current smoking, female gender, seasonal allergies, and "African American race", while having diabetes is protective (as reported in the Omapatrilat Cardiovascular Treatment versus Enalapril [OCTAVE] study). Her presentation was typical of bradykinin-dependent angioedema, namely, swelling lasting several days, without pruritus or urticaria, and being somewhat refractory to corticosteroids.

A15

Sneddon–Wilkinson subcorneal pustular dermatosis associated with an IgA monoclonal gammopathy

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Background: Subcorneal pustular dermatosis (SCPD) is a rare benign chronic inflammatory skin disorder of unknown etiology. First described by Sneddon and Wilkinson in 1956, it is characterized by a relapsing course of symmetric subcorneal sterile pustules involving the flexures, proximal limbs, and trunk. SCPD is often associated with a benign monoclonal IgA gammopathy, which can either precede or follow diagnosis. [1].
Case: A 56-year old Caucasian female presented to our outpatient immunology-dermatology clinic with a seven-year relapsing history of a mildly pruritic and irritating pustular skin eruption under the arms, breasts, and around the groin. Physical examination showed several pea-sized flaccid pustules on an erythematous base in the axillae, groin, and submammary regions (Fig. 2). The patient was otherwise well with no signs of systemic disease.

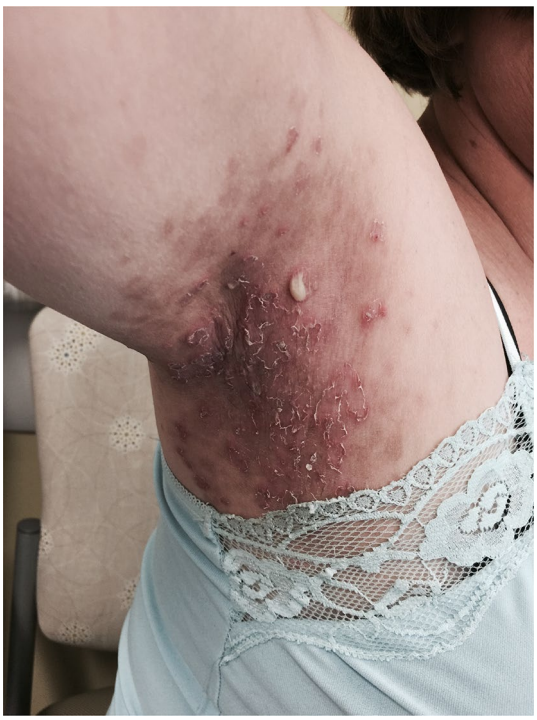

Fig. 2 Flaccid pustules measuring several millimetres in diameter on mildly erythematous skin. The *image* shows a classic half-and-half blister in which purulent fluid accumulates in the lower half of the blister

Investigations: Routine blood work including a complete blood count and liver function tests were normal. Serum protein electrophoresis showed an abnormal IgA monoclonal gammopathy lambda type with a value of 4.63 g/l (normal 0.7–3.52 g/l). Histopathology showed a sterile subcorneal pustule with inflammatory infiltrate of lymphocytes and neutrophils with the absence of acantholysis. Her previous treatments included topical corticosteroids and antibiotics as well as oral cephalexin and itraconazole, all with poor response.
Discussion: The main differential diagnosis of SCPD includes pustular psoriasis, pemphigus foliaceus, IgA pemphigus, impetigo, dermatitis herpetiformis, and acute generalized exanthematous pustulosis. Our patient had been previously treated with antibiotic and antifungal agents with poor response for an infectious cause of the disease. Clinicians may consider an inflammatory disease in their differential when presented with a pustular eruption in the interigenous areas.
Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Reference

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A16

Omalizumab can be effective in patients with allergic bronchopulmonary aspergillosis

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Background: Allergic bronchopulmonary aspergillosis (ABPA) is a challenging respiratory disease with significant morbidity and mortality. Among patients with severe asthma, 10 % have ABPA. Typically they have a decline in lung function, positive skin prick reaction to *Aspergillus*, elevated IgE levels, increased *Aspergillus fumigatus* specific IgE antibody and central bronchiectasis. Patients often exhibit refractory symptoms despite conventional asthma therapy. Long-term use of high dose inhaled and oral corticosteroids may lead to serious adverse effects such as immunosuppression, adrenal insufficiency, metabolic syndrome, hypokalemia, glaucoma, cataracts, peptic ulcers, osteoporosis, avascular necrosis, and psychiatric disturbances. Antifungals have many drug interactions and may require therapeutic monitoring. Some studies seem to suggest that omalizumab may be a better therapeutic option as there is some evidence demonstrating efficacy in ABPA and fewer adverse effects than long-term corticosteroids.
Methods: A retrospective chart review was performed for ABPA patients receiving omalizumab between 2007 and 2015 at our tertiary care Allergy and Asthma Clinic and The Ottawa Hospital Chest Clinic. Data was collected on demographics, total IgE, aspergillus specific skin testing, the use of inhaled and oral corticosteroids, quality of life (QoL) questionnaires, and number of asthma exacerbations/hospitalizations.
Results: A total of ten patients were evaluated (59.7 $\pm$ 13.9 years, 7 females and 3 males). Compared to their baseline, the average dose of inhaled corticosteroids dropped by 24 % at month 16 and out of the three patients who were dependent on oral corticosteroids, there was an 89 % decrease in prednisone use by month 16. QoL scores improved in relation to baseline. Nine of the subjects that reported exacerbations 12 months prior to treatment and none reported exacerbations once they started omalizumab.

Conclusion: Our retrospective study provides evidence to support the fact that omalizumab may be an effective low-risk treatment for patients with ABPA. Further prospective studies are required to confirm this finding.
Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

A17

Efficacious use of omalizumab in the treatment of cystic fibrosis

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Background: Cystic fibrosis (CF) is an autosomal recessive disease associated with airway obstruction and chronic lung infections. Allergic bronchopulmonary aspergillosis (ABPA) complicates 7–9 % of CF cases. The first-line therapies for treating ABPA are corticosteroids and antifungal drugs. Long-term use of corticosteroids may result in very serious adverse effects such as immunosuppression, adrenal insufficiency, and diabetes. Antifungals, mucolytics, pancreatic enzymes and bronchodilators are also other treatment modalities. A few studies suggest omalizumab may be effective in CF. Here we report a 25-year old male CF patient with concomitant ABPA complicated with numerous hospitalizations and steroid side effects who responded well to omalizumab.
Methods: A retrospective chart review was performed. Evaluation jointly occurred at The Ottawa Hospital's CF Clinic and a tertiary care Allergy and Asthma Clinic in Ottawa.
Results: The patient was diagnosed with CF at 5 months old. This patient had a positive skin prick reaction to *Aspergillus fumigatus* and a total serum IgE of 111.7 IU/mL. He developed corticosteroid-induced diabetes at age 19. Given his poor health and high IgE levels, a decision was made to attempt a trial of omalizumab. After 8 months of omalizumab, he successfully weaned off of prednisone. After 18 months, the patient no longer required insulin to treat his prednisone-induced diabetes. After 20 months, his quality of life improved. During the 12 months prior to treatment he was hospitalized 8 times. He had only one hospitalization since month seven. He was considered for a double lung transplant, but after 1 year of treatment, transplantation was no longer for consideration because he stopped needing daily home oxygen.
Conclusion: Omalizumab had a dramatic steroid sparing effect, reduced hospitalizations and O_2 requirements in this CF patient with ABPA and prednisone-induced diabetes. Further prospective studies using a larger cohort are necessary to make any clinical recommendations.
Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

A18

HAE with normal C1-INH with inconsistent response to C1 esterase inhibitor infusion but reliably responsive to icatibant

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Background: Hereditary angioedema (HAE) with normal C1-esterase inhibitor (C1-INH) is subdivided into factor XII mutation or of unknown origin (U-HAE). The diagnosis is based on clinical symptoms of recurrent edema (commonly skin swellings, tongue swelling), family history, and normal C1-INH quantity and function. U-HAE is presumed when factor XII mutation testing is negative. Here we present a 65 year old male with suspected U-HAE, who has a 30-year personal history of recurrent upper airway swelling, family history, and normal C1-INH quantity and function. He initially responds to empiric C1-INH. However, he seems to have developed resistance to C1-INH, requiring rescue therapy with icatibant.
Methods: For each treatment, a log method was used to collect information on: attack intensity, anatomical location, number of doses, onset of relief, time elapsed until complete resolution. Hospital records and patient-reports were collected for each treatment received through the emergency department.
Results: This patient was first seen September 2013 for suspected HAE. C4 and C1 INH quantities and function were repeatedly normal. In October 2013, the patient began IV treatment with CI-INH for the first time with good response. In June and October 2014, treatment with multiple doses of C1-INH was required to achieve resolution of symptoms. In February 2015, despite multiple C1-INH doses, the patient was intubated and admitted to ICU for tongue swelling with throat involvement, which resolved slowly over 4 days. In April 2015, icatibant was used for treatment in the ED when on tongue swelling occurred and was unresponsive to C1-INH. Swelling began to subside within 1 h of the icatibant administration. Since then, the patient has had many documented swellings that have not responded to C1 but have responded to icatibant.
Conclusion: Icatibant can be an effective treatment for suspected U-HAE when treatment response with intravenous C1 esterase inhibitor is inadequate.
Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

A19

Anaphylaxis reaction to lactase enzyme

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Background: Lactose intolerance affects approximately 20 % of Canadians and roughly 70 % of the world's population, leading to symptoms of flatulence and bloating after the ingestion of lactose-containing foods. Lactose intolerance develops primarily due to the absence of the enzyme lactase and can occur in childhood or adulthood. Treatment involves ingestion of commercially-available lactase enzyme preparations often produced by *Aspergillus* bacteria [1, 2]. Although anaphylactic reactions involving IgE mediated hypersensitivity have been reported for a number of foods, this case report represents the first documented evidence of anaphylaxis after exposure to supplemental lactase enzyme preparation.
Case report: A 38 year old white female teacher presented with a history of adult-onset lactose intolerance and a suspected allergy to lactase tablets after an episode of bilateral orbital swelling, shortness of breath, and throat constriction that responded to diphenhydramine. The patient's past medical history included oral allergy syndrome and medication-controlled asthma. She handled lactase tablets for years due to her children being lactose intolerant but only recently began using the tablets herself. Physical examination was benign, and skin prick testing to a slurry of the lactase tablet revealed a strongly positive reaction wheal size of 10 mm and flare of 60 mm with normal controls (Fig. 3). The patient required cetirizine treatment in clinic. Skin testing was performed with individual ingredients of the lactase tablet provided by the manufacturer and *Aspergillus niger*, a common bacteria used in lactase preparations. Only concentrated lactase enzyme elicited a positive response. Avoidance of lactase tablets was advised and the patient was educated in the use of injectable epinephrine.
Conclusion: This is the first documented case report of an anaphylactic reaction to lactase enzyme. The patient experienced systemic symptoms including shortness of breath and orbital swelling, reinforcing the importance of education and avoidance of triggers in these rare circumstances.
Disclosures: None.