on the basis of several factors, such as the length of exposure and duration of symptoms [2]. Treatment of hypersensitivity pneumonitis is based on antigen avoidance and immune suppression.
Case presentation: A 73-year-old female was admitted to hospital with a history of dyspnea and non-productive cough. Past medical history was significant for type-2 diabetes mellitus, osteoarthritis, and a 50-pack year smoking history. Social history revealed electronic cigarette use for smoking cessation. Physical examination was significant for a room air saturation of 82%, and bilateral inspiratory crackles. A CT chest revealed diffuse ground glass opacities in the upper lung fields, associated subplueral septal thickening, traction bronchiectasis, and areas of honeycombing. Bronchial washings did not yield positive culture results for bacterial, fungal, or mycobacterial agents. Screening autoimmune testing was negative, and a nasal swab sample was negative for viral agents. A diagnosis of chronic hypersensitivity pneumonitis relating to electronic cigarette use was made, and the patient was started on a tapering dose of prednisone. At a 1 month follow-up, the patient was noted to be symptom free, and chest radiography revealed significant interval improvements.
Conclusions: In the absence of recognized diagnostic criteria, hypersensitivity pneumonitis requires a high-index of suspicion for diagnosis. This case is among the first to demonstrate a link between electronic cigarettes and the development of hypersensitivity pneumonitis.
Statement of consent: Consent to publish was obtained from the patient involved in this study.

A82

Case report: can an insulin allergy be desensitized?

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Background: Allergies to Insulin are rare; the prevalence of having an allergy to insulin therapy is 0.1–3%. Immediate hypersensitivity reactions (type 1) are the most common and in rare cases can cause anaphylaxis. Insulin desensitization has been proven as an effective treatment for developing a tolerance to insulin.
Case presentation: R.S is a 68-year-old Caucasian male. He was diagnosed with type 2 Diabetes Mellitus 25 years ago and has a history of pruritus and urticaria after subcutaneous administration of various brands of premixed insulin. Skin prick testing revealed a positive reaction to Glulisine, Glargine, Detemir, Lispro, Novorapid, Novomix, and Aspart. His HbA1C at the time of skin testing was 10.2%. Based on the patient's history and recent hospitalizations he was determined to be an ideal candidate for rapid insulin desensitization. The insulin desensitization was performed in a controlled environment with a goal of reaching 50units of Glargine insulin. Overall the patient tolerated the desensitization very well with minor site reactions and pruritus. Presently the patient continues to tolerate 50units of Glargine qhs with Cetirizine for pruritus and has a HbA1C of 8%.
Conclusions: Rapid subcutaneous Insulin desensitization can be successful in treating patients with an Insulin allergy. Careful selection of patients for this procedure is necessary.
Statement of consent: Consent to publish was obtained from the patient involved in this study.

A83

Malignancy in the setting of hereditary angioedema (HAE): a possible consequence of chronically low C4 from C1-INH deficiency or dysfunction

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Background: HAE is a condition characterized by recurrent angioedema in the setting of deficiency of C1-inhibitor (C1-INH), which in turn leads to hyper-activation of kallikrein and overproduction of bradykinin. The lack of potent inhibition of C1r and C1 s by C1-INH results in chronically low/undetectable levels of C4. Previous reports have demonstrated increased risk of autoimmune disease and lymphoproliferative disorders in patients with congenital C4 deficiency [1]. This raises the possibility that low C4 in HAE may also predispose to these conditions. This may in turn influence the choice of therapy and treatment targets. We describe a case of a patient with HAE with chronically low C4 who went on to develop splenic B cell lymphoma.
Case presentation: A 52 yo F with recurrent episodes of acute abdominal pain since the age of 20 (with prior surgery for suspected diverticulitis) as well as a family history of angioedema in her mother was diagnosed with HAE after she presented with laryngeal edema requiring a tracheostomy. She was found to have an undetectable C4 of < 0.04 g/L (0.13–0.52 g/L) and a low C1 inhibitor level at < 0.03 (0.12–0.35 g/L). She went on to develop recurrent attacks of laryngeal and suspected bowel wall edema, initially treated with danazol, followed by IV C1-INH on demand treatment. Although her attacks reduced in frequency, her C4 levels remained chronically low/undetectable. At 70 years old, she was diagnosed with splenic B cell lymphoma with marrow involvement. Soon after she was admitted with sepsis and unfortunately died despite treatment.
Conclusions: As congenital C4 deficiency has been associated with an increased risk of malignancy, future consideration may need to be given to whether HAE with low C4 is best managed by C1-INH replacement at doses which restore C4 levels versus treatments, which only prevent kallikrein activation but do not restore C4 levels.
Statement of consent: Consent to publish was obtained from the patient involved in this study.

A84

A case of selective IgA deficiency: orchestrated by the microbiome?

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Background: Initially provided by breastmilk, secretory immunoglobulin A (sIgA) interacts with gut microbiota during infancy to shape gut mucosal immunity development. Individuals with selective IgA deficiency (SIgAD) have increased risk for sinopulmonary and gastrointestinal infections, atopy, asthma and autoimmune diseases [1–4]. Gut microbial dysbiosis from antibiotic treatment and/or limited breastfeeding may increase risk of these outcomes in IgA-deficient patients. Currently, SIgAD diagnostics and treatment are limited. This case discussion will focus on an IgA-deficient 4-year-old male seen at 18 months for failure to thrive and chronic sinopulmonary infection [3,5,6].
Case presentation: The patient presented with recurrent pneumonia, failure to thrive and intermittent respiratory distress. He was born early term with low birth weight and had trouble breastfeeding, prompting maternal treatment with the galactagogue, domperidone. Despite a suggestive history of dairy allergy, he now tolerates dairy. Workup demonstrated undetectable IgA and IgG at 6 months, with undetectable IgA and normal IgG at 18 months. Other immune system markers were normal and cystic fibrosis was ruled out. The patient had symptoms suggestive for asthma and responded well to inhaled corticosteroid and bronchodilator; though by 3 years asthma symptoms had resolved. The patient also had episodes consistent with pneumonia, which lessened in frequency and severity over time. By age 1, he had received 2 courses of clarithromycin and one of amoxicillin, all in succession.