dimethylamine (5.9%), gallate mix (4.5%), hexahydro-1,3,5-tris-(2-hydroxyethyl)triazine (2.1%), hydroabietyl alcohol (2.1%). The decrease of sensitisation to methylisothiazolinone (18.4% in 2014 and 2.0% in 2019, $P < .05$) was observed. The increase of sensitization to oleamidopropyl dimethylamine (5.3% in 2014 and 10.1% in 2019, $P < .05$) and gallate mix (0% in 2014 and 14.1% in 2019, $P < .05$) was observed. The prevalence of the other most common allergens was variable (statistically insignificant) or remained stable during the 6 year period.

Conclusion: In our study, the face was the most common body part for cosmetic ACD and female developed much more positive patch test reactions to cosmetic allergens than males. Changes of positive reactions to allergens over the years can reflect different exposure and changed composition of the cosmetics.

1240 | Autoimmunity and its clinical relevance in chronic urticaria - experience of a Portuguese UCARE center

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Background: Autoimmunity is thought to be one of the most frequent causes of chronic spontaneous urticaria (CSU).

Method: Retrospective analysis of CSU control (defined by a *Weekly Urticaria Activity Score* (UAS7) < 7), autoimmunity laboratory parameters (IgG anti-thyroperoxidase antibodies (anti-TPO), antinuclear antibodies (ANA), anti-dsDNA), total IgE, autologous serum skin test (ASST), basophil activation test (BAT) and its clinical relevance, from the patients observed in specialized urticaria consultation in a UCARE center in Lisbon, Portugal, from January 2017 to September 2019.

Results: 421 patients, 326 (77%) female, Average age 44.9 ± 16.7 years old. 75.8% had CSU, 14.2% Chronic inducible urticaria (CIU) and 10% CSU+CIU. We excluded patients with only CIU, so that 351 patients were included. The majority of them had controlled disease (n = 295; 84%). Due to lack of UAS7 registration, 6 patients were not characterized in controlled/ uncontrolled disease and others did not have all the evaluated parameters available (patients who lost follow-up/ missed urticaria consultation). In the controlled disease group, only 283 patients had laboratory values for total IgE (average value of 209.8kU/L), 263 patients for anti-TPO (positive in 28 (10.6%)), 256 for ANA (positive in 16 (6%)) and 236 for anti-dsDNA (positive in 4 (1.7%)); 67 ASST (positive in 24 (35.8%)) and 37 BAT (positive in 13 (35.1%)) performed. In the uncontrolled group (n = 50), all patients had laboratory values for total IgE (average 221.8kU/L) and for anti-TPO (positive in 2 (4%)), 49 patients had laboratory values for ANA (positive in 1) and 44 for anti-dsDNA (all negative); 13 ASST (positive in 3 (23%)) and 4 BAT (one positive) performed. In patients taking 4 anti-histamines/ day (n = 78), average total IgE was higher – 246 kU/L. Patients with positive ASST/BAT (n = 41) or ANA/anti-dsDNA (n = 21) had significant lower average levels of total IgE (173.3 kU/L) compared with those with negative tests (total IgE 235.6 kU/L). Regarding anti-TPO, in patients with positive ASST/BAT or ANA/anti-dsDNA, anti-TPO average level was higher (39.8 U/mL) than in patients with negative tests (average anti-TPO 21.1 U/mL).

Conclusion: In our population, we could not identify any laboratory parameter related to uncontrolled disease. CSU patients, with the present autoimmune parameters evaluated, had significant lower levels of total IgE and higher levels of anti-TPO, which is in line with literature.

1241 | Berotralstat (BCX7353) treatment demonstrates robust and durable reduction in the rate of hereditary angioedema (HAE) attacks over 48 weeks of the phase 3 APeX-2 study

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Background: Berotralstat is a novel oral once-daily highly selective plasma kallikrein inhibitor in development for HAE attack prophylaxis. Compared to placebo, berotralstat reduced HAE attack rates and was safe and generally well-tolerated in the 24-week, Phase 3 randomized double-blind placebo-controlled APeX-2 study (NCT03485911), with a favorable efficacy-safety balance.

Method: A total of 121 subjects with HAE Type 1/2 and ≥ 2 investigator-confirmed attacks in a 14-56 day baseline period were randomly assigned 1:1:1 to berotralstat 110 mg:150 mg: placebo for 24 weeks (6 months) and 108 subjects completed this part of the study (Part 1). At Week 24, subjects assigned to berotralstat 110 mg or 150 mg in Part 1 continued on the same dose for an additional 24 weeks and subjects assigned to placebo were re-randomized 1:1 in a blinded manner to receive berotralstat 150 mg or 110 mg (Part 2). Here we present results from preliminary analysis of long-term effectiveness of 150 mg berotralstat in Parts 1 and 2 of the ongoing APeX-2 study.

Results: The mean (±SD) baseline investigator confirmed attack rate for the 150-mg dose group intent to treat population (ITT, N = 40) was 3.06 (±1.56) attacks/month; the mean (±SD) attack rate declined to 1.72 (±1.73) in Month 1, 1.70 (±1.93) in Month 6, and 1.06 (±1.43) in Month 12. Median (range) attack rates declined from 2.70 attacks/

month (0.86-6.67) at baseline to 1.28 attacks/month (0.0-7.00) in Month 1, 0.97 (0.0-7.72) in Month 6, and 0.0 (0.0-4.00) in Month 12. Similar results were obtained in the 150 mg completers population (N = 30). Mean attack rates (±SD) for the completers population declined from 2.92 (±1.51) attacks/month at baseline to 1.43 (±1.45) at Month 1, 1.26 (±1.59) at Month 6 and 0.99 (±1.35) in Month 12. Median (range) attack rates declined from 2.44 attacks/month (0.86-6.59) at baseline to 1.00 (0.0-4.00) in Month 1, 0.97 (0.0-5.79) in Month 6, and 0.0(0.0-4.00) in Month 12.

Conclusion: Berotralstat 150 mg reduced mean and median monthly HAE attack rates as early as Month 1 of treatment. These reductions persisted and improved through Month 12 of treatment, with no evidence for development of tolerance to berotralstat. These findings suggest that long-term treatment with berotralstat 150 mg, an investigational once-a day oral therapy, provides sustained protection from HAE attacks and important benefits in HAE prophylaxis.

1258 | Foot allergic contact dermatitis to mercaptobenzothiazole, dipotassium dichromate, tetramethylthiuram disulfide (tmtd) with widespread involvement in periods of exacerbation

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Case Report: A 56-year-old girl presented with severe, bilateral foot dermatitis. Initially the lesions were localized in foot area and on the next day progressed to the buttocks and lumbar area. The patient was referred to the Outpatient Clinic and was treated with antihistamines, but her dermatological state deteriorated rapidly. At the day of admission to the Department of Emergency, numerous erythematous and vesicular lesions were present on the skin of the abdomen, thighs, and back, but the skin of the neck, chest, and extremities was also covered with erythematous and edematous patches. On the second day of hospitalization, we observed the evolution of lesions localized within the chest and extremities into an erythema multiforme-like targetoid eruption. Initially, the patient was treated with intravenous injections of dexamethasone and ceftriaxone and orally with second-generation antihistamines (in four-fold doses), followed by intravenous methylprednisolone pulse-therapy (total dose of 3 g). Diagnostic methods included: laboratory analyses (leukocytosis, neutrophilia, lymphopenia could be observed, and also serum CRP elevation). Histopathological examination revealed: massive edema of dermal papillae, leading to the formation of subepidermal vesicles; individual cell necrosis was observed in the upper epidermis. Within the dermis, a dense, perivascular inflammatory infiltrate was detected: The clinical picture suggested erythema multiforme. Three months after remission patch tests with standard and specific series was performed revealing a significant positive reaction to mercaptobenzothiazole ++, dipotassium dichromate +++, tetramethylthiuram disulfide (tmtd) + +. A thorough review of his history revealed that he was likely being exposed through her canvas sneakers. After implementation of allergen avoidance measures, his dermatitis resolved.

1260 | Factor XII hereditary angioedema: Clinical characterization of four families

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Background: Hereditary angioedema (HAE) is a rare genetic disease. In cases of HAE with normal levels and function of C1 inhibitor, the most frequent cause is due to Factor XII mutation (FXII-HAE).

Our aim is the characterization of HAE patients with normal complement and documented FXII mutation, followed in our Immunoallergology Department.

Method: Of the 129 patients with HAE followed in our out-patient Immunoallergology Department, we included all patients with genetic confirmation of factor XII mutation, by direct sequencing of exon 9 of the FXII gene. All patients also performed direct sequencing of exons 1-8 of the SERPING1 gene, to exclude mutations in this gene. We reviewed their clinical records and evaluated demographic, clinical and laboratorial data.

Results: We identified 6 patients with FXII-HAE of 4 different families: 2 from Portugal, 1 from Spain, and 1 with Portuguese and Egyptian ancestry. The 6 patients were all women, and had an average age of 36 ± 21 years (min 6, max 69). The 6 years-old child is still asymptomatic. Three developed spontaneous symptoms during adolescence; and two in their twenties in association with oral contraceptives.

Their average age at diagnosis was 33.3 ± 21.6 years of age (min 2, max 69), showing a delay up to 23.4 ± 14.7 years (min 3, max 44).

Triggers of exacerbations were: spontaneous in all, caused by oral contraceptive in 4 of the 5, and caused by mechanical trauma in 4 of the 5. Of the 2 who got pregnant, 1 had exacerbations during pregnancy.

Of the 5 symptomatic patients, the exacerbations affected: the face and hands in all 5, the feet in 4, the upper respiratory airways in 4, and the abdomen in 2 (with one undergoing unnecessary emergency appendectomy).

Three patients are medicated with anti-fibrinolytic drugs during exacerbations, with clinical improvement. Two were medicated with C1-inhibitor during exacerbations (with improvement).

Four patients, from 3 families, had the mutation p.Thr309Lys on exon 9 of FXII. The 2 patients, from the Spanish family, had a missense mutation g.6346 C>A on exon 9 of FXII.

Conclusion: In accordance with published data reports, our FXII-HAE patients show a female predominance. Exacerbations were