

EAACI Skin Allergy Club Zurich, Switzerland 8 – 9 June 2018

### Friday, 8 June 2018 EAACI Headquarters office

14:30 – 14:40	Welcome and introductory remarks Knut Brockow, Munich, Germany Charlotte Gotthard Mørtz, Odense, Denmark Martin Glatz, Zurich, Switzerland Pavel Kolkhir, Moscow, Russia
14:40 - 16:20	<ul> <li>Topics: Mastocytosis and atopic dermatitis</li> <li>Five oral presentations by JMs (15 minutes each + 25 minutes for discussion and coffee break) <ul> <li>"A case of the flushing patient" Caroline Weisser, Canada</li> <li>"KIT D816V mutation in peripheral blood of patients with mastocytosis in the skin" Polina Pyatilova, Russia</li> <li>"Diagnosis and follow-up of the mastocytosis: The experience of an Italian centre" Claudia Bussolino, Italy</li> <li>"Two Cases of Adult Onset Urticaria Pigmentosa with the Final Diagnosis of Indolent Systemic Mastocytosis" Ceyda Tunakan Dalgic, Turkey</li> <li>"Natural Moisturizing Factor as a clinical marker for severity of atopic dermatitis" Anouk Eva Marina Nouwen, The Netherlands</li> </ul> </li> </ul>
16:20 - 16:40	Lecture: "Allergic reactions in mastocytosis" Prof. Knut Brockow, Munich, Germany
16:40 - 17:00	Lecture: "Atopic dermatitis or allergic contact dermatitis – how to differentiate?" Prof. Charlotte Gotthard Mørtz, Odense, Denmark
17:00 - 19:45	Free time
19:45	Meeting in the hotel lobby & walking to the restaurant
20:00	Dinner at the Restaurant Riedbach



### Saturday, 9 June Dermatology Department, University Clinic Zurich

07:45 – 08:30 Breakfast at the Hotel Ibis

# 09:00 – 10:45 Topics: Contact dermatitis, atopic dermatitis, urticaria and eczema

*Five oral presentations by JMs* (15 minutes each + 30 minutes for discussion)

- "Associations of early life environmental and genetic factors with eczema phenotypes. The Generation R study" Chen Hu, The Netherlands
- "In Vivo Confocal Laser Scanning Microscopy in Autoimmune Bullous Dermatoses and Acute Allergic and Irritant Contact Dermatitis"

Anastasiia Allenova, Russia

- "Sensitivity to Topical Testosterone Formulations" Stephanie Vakaljan, Canada
- "Fixed drug eruption associated with pseudoephedrine a case report"
   Olga Branicka, Poland
- "Updosing of desloratadine (10mg) in chronic spontaneous urticaria"
   Gauri Godse, India
- 10:45 11:00 Coffee break

 11:00 - 11:30
 Lecture: "Biologics for atopic dermatitis - is the topical treatment obsolete?"

 Dr. Martin Glatz, Zurich, Switzerland

11:30 – 12.30 Group Visit Department of Dermatology University Clinic in Zurich (led by Dr. Martin Glatz)



### Session 1 Friday 8 June 2018 EAACI Headquarters

#### 1. A case of the flushing patient

<u>Caroline Weisser</u><sup>1</sup> <sup>1</sup>Hospital for Sick Children, University of Toronto, Canada

Cutaneous flushing is a common presentation to allergists, dermatologists and other allied health practitioners. Flushing results from changes in cutaneous blood flow triggered by multiple conditions and fortunately, most cases are caused by benign disorders. Taking a thorough history and physical examination helps elucidate these conditions, and in some cases, further laboratory, radiologic and histopathologic studies are required to differentiate more serious diagnoses. In particular, carcinoid syndrome, pheochromocytoma, mastocytosis and endocrine tumours need to be considered. This case reveals the difficulty in making a diagnosis in a patient with prior history of cutaneous mastocytosis who is now exhibiting systemic symptoms including episodic flushing. The approach will analyze and help establish an algorithm, diagnosis and management of conditions that produce flushing.

## 2. **KIT D816V** mutation in peripheral blood of patients with mastocytosis in the skin

<u>Pyatilova P.M.</u><sup>1</sup>, Teplyuk N.P.<sup>1</sup>, Olisova O.Yu.<sup>1</sup>, Lukina K.A.<sup>2</sup>, Kovrigina A.M.<sup>3</sup> and Dukhanin A.S.<sup>4</sup>

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**Background**: Mastocytosis is a heterogeneous group of disorders associated with excessive proliferation of atypical mast cells in skin and/or internal organs. The KIT D816V mutation is considered to be one of the main reasons of disease development and is diagnosed in almost all patients with systemic process. However, the prognostic and diagnostic value of this mutation remains controversial due to the sensitivity of used methods, type of samples (bone marrow, peripheral blood, skin), mast cell burden and involvement of other lineages.



**Methods**: 20 adult patients with mastocytosis were examined. The KIT D816V allele burden was measured by allele-specific quantitative PCR assay (ASOqPCR) in peripheral blood (PB) of 12 patients. Additional studies included bone marrow investigation, complete blood count, detailed biochemical blood investigation, serum tryptase (sT) measurement, ultrasound of abdomen, KIT D816 mutation testing of PB by direct sequencing, osteodensitometry or x-ray of bones and gastrointestinal tract investigation.

**Results**: All patients presented with urticaria pigmentosa lesions (male, n=5; female, n=15; mean age, 42; range, 20-66). According to the WHO criteria they were classified to cutaneous mastocytosis (CM, n=2, 17%), indolent systemic mastocytosis (ISM, n=4, 33%), smoldering systemic mastocytosis (SSM, n=3, 25%), aggressive systemic mastocytosis (ASM, n=3, 25%). 18 patients didn't undergo bone marrow examination and were pre-diagnosed with mastocytosis in the skin (MIS). 10 (83%) of 12 patients, diagnosed with CM (n=2), ISM (n=1), ASM (n=1) and MIS (n=6), showed positive KIT D816V mutation in PB samples detected by ASOqPCR. In contrast, the KIT D816V mutation was diagnosed in 1 (8%) of 12 cases by direct sequencing assay. Percentage of KIT D816V mutation-positive cells varies from 0,7 to 61 (mean=19) and did not correlate with serum tryptase levels. In 6 (50%) of 12 patients with sT levels <20ng/mL KIT D816V mutation was detected by ASOqPCR with mean allele burden 14% (range, 0,7-48%). Interestingly, 2 cases with high sT levels (57ng/mL, 119ng/mL) tested negative.

**Conclusions**: KIT D816V mutation status examination in PB samples by ASOqPCR is a useful and sensitive method in the diagnostic work-up of mastocytosis patients. However, further studies are required to determine the prognostic importance of this test and its correlation with different types of the disease.

# 3. Diagnosis and follow-up of the Mastocytosis: The experience of an Italian centre

#### <u>Claudia Bussolino<sup>1</sup></u>, Giovanni Rolla<sup>1</sup>

<sup>1</sup>Division of Allergy and Clinical Immunology of Mauriziano Umberto I Hospital, Torino, Italy

**Background and Objectives:** Mastocytosis, a type of mast cell disease, includes a broad spectrum of various clinical entities very heterogeneous by symptomatology, course and prognosis. Therefore, there is a need to standardize diagnostic and prognostic criteria and therapeutic protocols.

**Methods**: All the cases of anaphylaxis diagnosed in the Division of Allergy and Clinical Immunology of Mauriziano Umberto I Hospital have been analyzed. 17 patients were affected by cutaneous mastocytosis (CM) and 15 by systemic mastocytosis (SM). The serum tryptase was measured in all patients. We diagnosed CM or SM on the basis of the WHO-criteria.



We indicated the execution of bone marrow biopsy in all patients with suspected SM and tryptase level >20 mg/L. In patients with tryptase level  $\leq$  20 mg/L and without signs of systemic involvement, we repeated the measurement of serum tryptase every 6 months, to observe any increase. Every patient with diagnosis of SM was pointed out to follow-up, six-monthly measurement of serum tryptase, annual abdominal ultrasound and bone densitometry.

**Results:** There was a prevalence of CM in children (average age 28 years). The average age of patients with SM was 53 years.

Among patients with CM, the cutaneous involvement was with Urticaria Pigmentosa (n=15) or with Solitary Mastocytoma (n=2). Among patients with SM: 13 had the indolent form (ISM), 2 the aggressive form (ASM). Two patients had SM associated to a myeloproliferative disorder: ISM+polycythemia vera, ASM+thrombocythemia JAK2+. One patient was affected by ISM associated to Mantle Cell Lymphoma. Patients with CM and ISM had mainly mediatordependent symptoms (flushing, urticaria, itching). Patients with ASM had either mediator-dependent and organ infiltration symptoms: urticaria pigmentosa (n=6), flushing and itching (n=8), hypotension (n=4), anaphylaxis (n=2), headache (n=4), myalgia (n=1), gastrointestinal symptoms (n=6), hepato/splenomegaly (n=1), osteopenia/osteoporosis/osteosclerosis (n=9). Patients with CM and SM were mainly in therapy with anti-mediator and membrane stabilizer drugs. Two patients with ASM were also in therapy with imatinib or IFN-a. The patient with polycitemia vera associated to ISM was in therapy with hydroxyurea. Four patients with SM and osteoporosis were in therapy with bisphosphonates. Patients with previous anaphylaxis had adrenaline-auto-injector, to be used in case of need. The serum tryptase levels were normal (< 20  $\mu$ g/L) in 9/17 patients with CM (average value 20,67  $\mu$ g/L). Among patients with SM, the serum tryptase levels were high in 14/15 (average value 100,06  $\mu$ g/L). We noticed higher serum tryptase levels in patients with ASM versus patients with ISM (average value 477  $\mu$ g/L versus 58  $\mu$ g/L). Patients that have received cytoreductive therapy (IFN-a, imatinib), had serum tryptase levels lowered of about 50% if responsive.

**Discussion and Conclusions**: The follow-up of patients with CM was about 8 years. In all of them, serum tryptase levels were constant over time and the clinical manifestations remained mainly cutaneous, confirming a low risk of evolution of CM in SM. The follow-up of patients with SM was about 4 years, none of them evolved from indolent to aggressive forms. In line with the literature, these data show that patients with ISM have, in the majority of cases, a good prognosis and life expectancy equal to general population<sup>1</sup>. The serum tryptase permits to discriminate from patients with CM and SM, and it is an useful marker for monitor the clinical progression of the variants of ASM. Our data confirm that it is important for: a) screening and differential diagnosis of SM in patients without skin localization; b) assessment of the evolution of CM in SM and of ISM in ASM; c) evaluation of the responsiveness to therapy<sup>2</sup>.



Regarding therapeutic protocols, the treatment is mainly symptomatic, with a good control and prognosis in patients with CM and ISM. Instead, ASM patients require a specific treatment with cytostatic agents, as IFNa, that are unfortunately effective only in a limited number of patients. Recently, the tyrosine kinase inhibitors showed better performances in the induction of remission<sup>3</sup>.

#### References

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2) Lawrence B Schwartz. Clinical utility of tryptase levels in systemic mastocytosis and associated hematologic disorders. Leukemia research, 25(7):553–562, 2001.

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4. Two Cases of Adult Onset Urticaria Pigmentosa with the Final Diagnosis of Indolent Systemic Mastocytosis

<u>Ceyda Tunakan Dalgıç</u><sup>1</sup>, Okan Gülbahar<sup>1</sup>, Emine Nihal Mete Gökmen<sup>1</sup> <sup>1</sup>Ege University Medical Faculty, Department of Internal Medicine, Division of Allergy and Clinical Immunology

**Introduction**: Mastocytosis is a disorder characterized by mast cell accumulation, commonly in the skin, bone marrow, gastrointestinal tract, liver, spleen, and lymphatic tissues. The World Health Organization divides cutaneous mastocytosis into three main presentations as mastocytoma, urticaria pigmentosa and diffuse cutaneous involvement. Urticaria pigmentosa is the most common cutaneous mastocytosis in children, and it can form in adults as well. It is thought to be a benign, self-resolving condition that remits in adolescence. Unlike child onset forms, urticaria pigmentosa is usually seen with internal organ involvement in adults. We report two female cases with adult onset monomorphic cutaneous mastocytosis with the final diagnosis as smoldering systemic mastocytosis.

**Case report 1**: 29-year-old woman was presented to our clinic with brownish macular rashes which had been present for 10 years. They had developed over her arms and progressed to her torso and abdomen during recent years. Between 2014 and 2015, during her pregnancy her brownish macular lesions were progressed, and finally these lesions have been progressed to her torso, abdomen and finally to her neck and face since the last 6 months (Picture 1). She described her lesions as erythematous and pruritic when scratched (Darier's Sign) and reported that it became irritated and erythematous during exercise, with exposure to heat, humidity and trauma.



The patient had hypotensive presyncope/syncope attacks which last for 5 to 10 minutes, accompanying with nausea, vomitting and headache for 2 years. 7 years ago, when she had been stung by a bee in the forehead, she had uterin contractions and bleeding, fecal incontinans, broncospazm, and finally she had a presyncope attack in 15 minutes. The patient had flushing attacks and besides that, she had back and hip pain for 3 years. In december 2017, the patient was eventually referred to our clinic for her typical monomorphic small maculopapular cutaneous lesions compatible with urticaria pigmentosa. Skin biopsy showed increased perivascular and interstitial mast cell proliferation, the histological stain revealed more than 25 mast cells expressing CD117 per high-power field. Serum baseline tryptase was 22.5 ng/mL. Bone marrow aspiration biopsy showed 60% cellularity; more than 15 spindle-shaped and oval mast cells in four focuses showing aberrant expression of CD117, CD25 and mast cell tryptase markers. Result of the bone marrow biopsy was compatible with systemic mastocytosis (SM). Ketotifen was prescribed for maintanence therapy.

Besides that, the patient had end-organ infiltration findings such as hepatosplenomegaly (spleen:138 mm, liver: 130 mm in abdominal ultrasound), cervical lymphadenopathy with the diameter of 2 cm, and lomber osteopenia. These findings are referred to as B signs (indicating high burden of mast cell expansion) that the patient was consulted to hematologist, and the final diagnose was indolent SM; but also the patient should be followed carefully for the high risk of developing smoldering SM.

**Case report 2**: 58-year-old woman had realised brownish macular rashes on her abdomen and torso in 1982 at first. She described her lesions as erythematous and pruritic when scratched (Darier's Sign) and reported that it became irritated and erythematous with exposure to heat and trauma. The rash involved mostly the patient's torso, abdomen, proximal extremities, and neck although her hands, feet, head and distal extremities were spared (Picture 2). In 2000, she was referred to dermatology clinic due to maculopapular cutaneous lesions. Skin biopsy revealed increased perivascular and interstitial mast cell proliferation, the histological stain revealed 32 mast cells expressing CD117 per high-power field, diagnosed as urticaria pigmentosa. Serum baseline tryptase was 56 ng/mL. Between 1998 and 2004 years, she had presyncope/syncope attacks lasting approximately 30 minutes without any precipitating factors. Since 2010, she also had flush attacks without any precipitating factors. In 2010, bone marrow aspiration biopsy was performed. Biopsy showed 80% cellularity; aberrant expression of CD117, CD25 and mast cell tryptase markers were detected on spindle-shaped and oval mast cell groups with more than 15 mast cells in two focuses. The result was compatible with systemic mastocytosis. Peripheric blood molecular studies showed presence of codon D816V mutation on myeloid multileage cells and mast cells. Given the patient's symptoms, persistently elevated tryptase, mast cell proliferation on skin and bone marrow biopsy, and the presence of KIT D816V mutation, she met the criteria for the diagnosis of systemic mastocytosis (SM).



Ketotifen was prescribed for maintanence therapy. Besides them, the patient had end-organ infiltration findings such as hepatosplenomegaly (spleen:150 mm, liver: 170 mm in abdominal ultrasound), and diserytropoesis in bone marrow biopsy. Interestingly, the patient was diagnosed with REM sleep behaviour disorder in 2015; that was evaluated due to mast cell infiltration of central nervous system. These findings are referred to as B signs that the patient was consulted to hematologist, and the final diagnose was indolent SM; but also the patient should be followed carefully for the high risk of developing smoldering SM.

**Discussion and Conclusion**: Systemic mastocytosis is a rare disease process driven by aberrant activation and infiltration of mast cells that presents in adults with nonspecific symptoms and can follow an indolent or aggressive course. In adults, the most common form is ISM, as seen in the patients presented above. Clinicians need to keep a high index of suspicion in order to identify patients with SM due to the low prevalence of disease and nonspecific symptoms. Referral to hematology should be made in severely symptomatic patients and those with high pretest probability for systemic disease.



Picture 1 (left): Typical monomorphic small maculopapular cutaneous lesions (urticaria pigmentosa) are seen on the skin of the patient's shoulder. Picture 2 (right): Typical monomorphic small maculopapular cutaneous lesions (urticaria pigmentosa) are seen on the skin of the patient's abdomen.



## 5. Natural Moisturizing Factor as a clinical marker for severity of atopic dermatitis

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**Introduction:** A close correlation has been found between decreased Natural Moisturizing Factor (NMF) concentrations in the stratum corneum and the presence of loss-of-function mutations in the pro-filaggrin gene (FLG). Filaggrin plays an important role in establishing the barrier function of the skin. FLG-mutations are the strongest genetic risk factor for the development of Atopic Dermatitis (AD) and are associated with early-onset and a more severe disease course. An estimated 30-50% of AD-patients have a FLG-mutation. The NMF content in the stratum corneum can be rapidly and non-invasively measured by Raman spectroscopy. To date NMF has not been used for clinical stratification of AD-patients. Our aim was to examine the association of clinically measured NMF values with disease severity of AD, early onset of AD  $\leq$  6 months, food allergy, asthma, and allergic rhinitis in children.

**Methods:** We conducted a retrospective, single-center study in AD patients in the age-range of 0-to-17 years. NMF levels in the palmar stratum corneum were measured in vivo using Raman spectroscopy. Clinical parameters were obtained retrospectively from electronic patient files. Univariate and multivariate logistic regression models were used to examine the associations between NMF content and the clinical parameters.

**Result:** 209 patients were included in the analysis for binary NMF values (decreased/normal). In the univariate logistic regression models decreased NMF values were associated with increased odds for severe AD, 2.30 (1.20, 4.41), sensitization for food allergens, 2.21 (1.22, 3.99), sensitization for inhalation allergens, 2.37 (1.31, 4.29) and food allergy, 3.05 (1.50, 6.23). These associations remained significant when adjusted for gender and age during NMF measurement.

**Discussion:** The strong observed association of NMF levels in the stratum corneum with AD severity and with other clinical disease parameters are an important step towards an objective clinical stratifier for patients with AD, using in vivo Raman spectroscopy.



### Session 2 Saturday 9 June 2018 Dept. of Dermatology, University of Zurich

# **1.** Associations of early life environmental and genetic factors with eczema phenotypes. The Generation R study.

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**Background and aim** Childhood eczema is highly prevalent, but variable in onset and persistence of which specific early life environmental and genetic factors are not fully known. We aimed to identify eczema phenotypes during childhood, and the associations of early life environmental and genetic factors with these identified phenotypes.

**Methods** In this study among 5,297 children of a multi-ethnic population-based prospective cohort study, latent class growth analysis was applied to identify phenotypes based on parental-reported physician-diagnosed eczema longitudinally measured from birth until age 10 years. Information on early life environmental exposures was obtained by questionnaires from birth until age 6 years. FLG genotype (≥1 of 4 most common mutations) and a genetic risk score based on 30 previously identified genetic variants were determined. Weighted adjusted multinomial models were used for association analyses.

**Results** We identified five eczema phenotypes including never (76%), early-(8%), mid- (6%) and late-preschool-onset-resolving (8%), and persistent eczema (2%). Nulliparity, parental history of atopic diseases, late-onset and persistent wheezing were most strongly associated with increased risks of early-preschool-onset-resolving (odds ratio (95% confidence interval):



range 1.37 (1.07,1.74) to 2.68 (1.95,3.70)) and persistent eczema (range 1.65 (1.06,2.55) to 3.38(1.95, 5.85)). Male sex was associated with an increased risk of early-preschool-onset-resolving eczema only (1.49 (1.18,1.89), and non-European ethnicity with late-preschool-onset-resolving (1.35 (1.03,1.78)) and persistent eczema (1.76 (1.10, 2.82)). Most effect estimates did not materially change when we additionally adjusted for genetic factors. The FLG genotype was associated with increased risks of early- (2.21 (1.39, 3.50)) and late-preschool-onset-resolving eczema (2.02 (1.26,3.24)), while the genetic risk score, per additional risk allele, was with late-preschool-onset-resolving (1.07 (1.02,1.12) and persistent eczema (1.08 (1.00,1.18)). We observed no associations of maternal education, breastfeeding, daycare attendance or pet exposure with eczema phenotypes.

**Conclusion** Five eczema phenotypes during childhood with differently associated early life environmental and genetic factors were identified. Our results may be useful for predicting eczema trajectories and potential preventive strategies.

2. In Vivo Confocal Laser Scanning Microscopy in Autoimmune Bullous Dermatoses and Acute Allergic and Irritant Contact Dermatitis <u>Anastasiia Allenova<sup>1</sup></u>, Natalya Teplyuk<sup>1</sup>, Ekaterina Shadrina<sup>1</sup>, Sergey Tkachenko<sup>2</sup> <sup>1</sup>I.M. Sechenov First Moscow State Medical University, Division of Immunemediated Skin Diseases, Moscow, Russian Federation <sup>2</sup>Russian Medical Academy of Continuing Professional Education, Moscow, Russian Federation

In vivo confocal laser scanning microscopy (CLSM) represents a new non-invasive high-definition imaging technique. CLSM allows rapid and real-time investigations of various skin structures at cytological resolution at the patient's bedside. To date CLSM is widely used to diagnose and monitor treatment of skin tumors and some inflammatory dermatoses. However, its capabilities in autoimmune bullous dermatoses (ABD) and acute allergic and irritant contact dermatitis are studied insufficiently, no systematic studies are available.

Autoimmune bullous dermatoses have severe course, frequent complications, a high mortality risk rate, and significant difficulties in differential diagnosis. Currently, there are several methods, namely histology, direct

immunofluorescent assay, and serological tests. Non-invasiveness of CLSM is important for ABD patients. Examination with CLSM has a great advantage of no tissue damage (the damage can be a trigger factor for a new bullas), quickly obtaining of the results, the possibility to identify acantholytic cells (AC) and significant comfort for the patient. CLSM is useful for differential diagnosis of common vesiculobullous dermatoses: acute allergic and irritant contact dermatitis. The clinical presentation may vary depending on the patient's reactivity, and triggering agents. However, each disease has a few typical morphological characteristics detectable by CLSM.



We observed 56 patients including 26 patients with pemphigus vulgaris (PV), 14 patients with bullous pemphigoid (BP), 7 patients with Hailey-Hailey disease (HHD), 9 patients with allergic and irritant contact dermatitis. All of them underwent CLSM and standard methods including skin biopsy. In ABD patients CLSM detected the main signs such as blistering levels, the presence of AC, dilated vessels, and inflammatory infiltration. The correlation between the CLSM findings and the histological examination results was evaluated. The diagnosis made by CLSM corresponded to the histological data in 88.5% (PV), 78.6% (BP) and 85.7% (HHD). Moreover, specific CLSM signs characteristic for ABD were revealed in the surrounding areas. This suggests the possibility of the disease progression assessment by identifying specific signs in unaffected skin. The main confocal findings also corresponded to histopathology in contact dermatitis patients. CLSM detected specific and nonspecific signs: blisters, inflammatory cells, spongiosis, epidermal necrosis, pleomorphic ballooned keratinocytes and some others. These main features made it possible to distinguish acute allergic or irritant contact dermatitis in all 9 patients. Thus, CLSM in vivo can be used not only as a tool which provides additional morphological data, though even as a method for replacing a traditional morphological examination. In this regard, further research is needed in this field.

#### 3. Sensitivity to Topical Testosterone Formulations

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#### Case Report:

A 36 year old male presented with query adverse reaction to topical testosterone versus irritant/contact dermatitis. The patient required supplemental testosterone following a urological procedure. Axiron cream was started by his GP, resulting in local skin reactions. The patient was then trialed on a patch formulation, Androtop, and complained of localized itchy, red, raised skin over areas where the patch was applied. It was his GP's preference to remain on a topical formulation due to the bioavailability of testosterone.

The patient was patch tested to the NACDG common 70 allergens, as well as samples of Axiron, Androgel, and Androderm, alternate patch and gel formulations. Additional allergens relevant to the testosterone formulations were added, including Sodium Benzoate and Povidone Iodine. There were also areas of skin patch tested that were pre-treated with hydrocortisone cream, with then samples of Axiron, Androgel, and Androderm applied on top, because of recommendations in the literature which cite some success with topical corticosteroid pretreatment.

Patch test readings at 72 hours revealed positives to Formaldehyde, as well as the Testaderm and Androderm testosterone formulations, including those pretreated with hydrocortisone. Androgel tested negative. The patient then began a trial of Androgel.



Four days into treatment, the patient developed a burning, red rash on the areas applied (shoulders) and discontinued Androgel therapy. Due to there being no alternate topical preparations available to test, injectable testosterone therapy was recommended. The first two injections were administered in office in a stepwise process, with 1/10 of the dose injected intramuscularly followed by the full dose. The injections were tolerated with no immediate or delayed reactions.

**Discussion**: While there are no formal testing guidelines currently for patch testing work up of topical hormone formulations, one may consider the utility of attempting pre-treatment with corticosteroids to see whether the patch/gel formulation is better tolerated. Further study is required to determine whether alternative topical therapies may be indicated, or an alternate route of therapy.

#### **4. Fixed drug eruption associated with pseudoephedrine – a case report.** <u>Olga Branicka<sup>1</sup></u>, Joanna Glück<sup>1</sup>

<sup>1</sup>Clinical Department of Internal Diseases, Allergology and Clinical Immunology, Medical University of Silesia, Katowice

**Introduction**: Fixed drug eruption (FDE) is often a missed diagnosis due to lack of characteristics of more common morbilliform drug rash. FDE is a circular, erythematous plaque that recurs as one or a few lesions always in fixed locations upon ingestion of a drug. The diagnosis can be confirmed by detailed interview about used medicines, patch tests and oral provocation methods with suspected drugs.

**Case report**: A 30-years-old man presented limited erythematous, and edematous plaques, always in the same places - axillary and inguinal cavities. The lesions appeared a few hours after taking the combined preparations containing pesudoephedrine. The patient also admitted that lesions were more severe after higher doses of pseudoephedrine, resolved within a few days, usually did not require medical intervention, usually he used calcium and antihistaminines. During allergological works-up patch tests with pseudoephedrine (SUDAFED) prepared in 10% petrolatum and 0,9% sodium chloride on the skin in the presence of skin lesions and in places where there were no changes were performed. Positive results were obtained both after 48 and 72 hours with both vehiculum and in both places.

**Conclusion**: Pseudoephedrine is often used in the course of upper respiratory tract infections to reduce the symptoms. In our case episodes of FDE can be attributed to pseudoephedrine based on the medical history and patch tests. The patient should be informed not to use the culprit drug and in the case of appearance of FDE beacuse of mistakenly drug administration anitihistamines may be used to reduce the symptoms of itch



### 5. Updosing of desloratadine (10mg) in chronic spontaneous urticaria <u>Gauri Godse<sup>1</sup></u>, Kiran Godse<sup>1</sup> <sup>1</sup>Shree Skin Centre, Nerul, Navi Mumbai India

**Introduction**: In India desloratadine 10 mg tablet is now available for treatment of skin allergies. We tried this formulation for treatment of chronic spontaneous urticarial in adult patients.

**Materials and methods**: Thirty three adults (11males 22 females) between age of 14 and 70 (average age 31.2 years) with chronic urticaria of more than 6 weeks duration (average duration 70 days) were included in this study. Basic investigations like CBC.TSH CRP and sugar were done to rule out focus of infection. Urticaria activity score was noted every week. All patients were started with desloratadine 10 mg per day and were reviewed every week for 4 weeks. Those patients who did not respond were up dosed with 20 mg of desloratadine (2 tablets) per day in two divided doses. Out of 33 patients 21 were under control with 10 mg per day and 9 were controlled with 20 mg per day. Three patients were advised cyclosporine for control of urticaria. Three patients complained of sedation and headache at 20 mg dose.

**Results and conclusion**: Out of 33 patients 21 and 9 patients responded to desloratadine 10 and 20 mg dose respectively. UAS score came down to less than 6 in most patients. Two patients were found to have hypothyroidism and three patients with anemia were appropriately treated. New 10 mg tablet of desloratadine was found to be suitable for treatment of urticaria with the advantage of less pill burden and less sedation.