Cocamidopropyl betaine may be preserved with Methylisothiazolinone, but this is not always declared on the final product.

Conclusion: The presence of skin sensitizing additives and impurities in raw materials used for cosmetic products poses a significant concern, since these are not always declared on the product or in the Material Safety Data Sheet (MSDS), it is crucial to obtain a comprehensive understanding of the raw material composition by requesting documents such as Technical data sheets (TDS), Product dossier, and 100% composition breakdown. Through this approach, specific examples such as Benzyl alcohol as an impurity in Candelilla cera and Benzyl alcohol and Benzyl benzoate in C12-15 Alkyl benzoate have been identified as potent triggers for perfume allergy.

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100480 | Cheilitis caused by contact allergy to toothpaste

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Allergic contact dermatitis (ACD) is an immunoinflammatory disorder caused by an antigen-specific T cell-mediated, delayed type hypersensitivity reaction elicited by the contact exposure of an allergen with the skin in a subject who has been previously sensitized to that specific allergen. The clinical presentation may vary depending upon the nature and concentration of the allergen and on the period of exposure, presenting as acute, subacute or chronic dermatitis, and in most cases, ACD lesions are primarily confined to the site of contact.Identifying the culprit agent may be challenging, but the offending allergen identification is important, since allergen contact avoidance is the key to preventing recurrence.

A 52-year-old female patient presented with a history of two months of recurrent episodes of lips swelling with small blisters and erythematous skin on her upper lip. Symptoms were well controlled by systemic corticosteroids and antihistaminic administration, however discontinuation of the therapy resulted in relapse of the dermatitis. Past medical history was unremarkable apart from hypothyroidism and she did not recall any changes in her hygienic or cosmetic routine in the last months, except for the switching to a new toothpaste. Since the morphology, regional distribution and temporal course of the clinical manifestations suggested ACS, patch tests were performed on the upper back with a dental (metal) series. Patch test readings were performed at day 2 and day 3. On day 2 no reactions were observed to any of the tested aptens, whereas at day 3 the patient showed a positive reaction to stannous oxalate (++) and sodium tetrachloropalladate (++).

Examination of all the hygienic and cosmetic products ingredients revealed the presence of stannous fluoride in the toothpaste (Sensodyne Sensitivity®) the patient was using, while no Nickel was in it. Consequently first, a semi-open test was performed using a 1% concentration of the suspected toothpaste with a 48 and 72-h reading, which resulted in a negative outcome. Next we conducted an open test using toothpaste "as is" (concentration 100%) to exclude an irritative cause. The toothpaste was removed after 24 h, and the result was negative. Finally, we performed a patch test using dilutions of 5, 10, 20, and 30 percent in water. At the 48-h reading, all dilutions yielded negative results. However, at the 3- and 7-day readings, the 20 and 30 percent dilutions produced positive results, with increasing positivity on day 7, indicating a delayed-type allergic reaction.

Following replacement of the toothpaste with a stannous fluoride-free product the stomatitis resolved and did not recur. Considering the clinical presentation, the patch test results and the resolution of symptoms after the removal of the suspected causative allergen, a diagnosis of allergic contact stomatitis was made. Contact allergies to toothpaste and its ingredients have already been described, however these have been previously assessed only to a very limited extent. With this case report we aim to highlight a potential cause of allergic contact dermatitis in which the culprit allergen could be under-recognized.

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100496 | The coexistence of psoriasis and allergic dermatitis: A case report

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Introduction: Correct diagnosis of hand dermatosis can be challenging. The medical history, clinical presentation, duration and course of the disease, relation to work, hobbies and co-morbidities, as well as concomitant medications should be considered. Positive history of psoriasis does not exclude the coexistence of contact dermatitis, atopy, or irritation. [Kolesnik M et al, 2018, Eczema in Psoriatico: An Important Differential Diagnosis Between Chronic Allergic Contact Dermatitis and Psoriasis in Palmoplantar Localization].

Case presentation: A 24-year-old female patient presented to our clinic with complaints of hyperemia, itching, burning sensation of the hands. These complaints first appeared 6 months ago. She had no history of similar problems. A little over a year ago the patient started working as a pastry cook. The patient does not smoke, is slightly overweight BMI 26.4, no comorbidities and does not take any medications regularly.

A month after the onset of her complaints, she consulted an allergist. The clinical picture and the investigations performed were the basis for the diagnosis of allergic contact dermatitis: total IgE 264 IU/mL in blood tests (ref. value <94), strong reaction to Vespula vulgaris (wasp) in the specific IgE panel; clear reaction to thymol; weak reaction to bee venom; rye; egg white and yolk. Diagnostic patch test not

performed. The patient was advised on hand protection, skin care and prescribed antihistamines. Despite treatment and being on sick leave, the above complaints persisted.

In March this year, the patient was referred to the Dermatology Clinic, where we performed a skin punch biopsy, fungal examination, and microbiological culture test. Microscopically, no fungal structures were found, *Staphylacoccus aureus* and *Candida sp.* were isolated. Histologically, an irregularly thickened parakeratotic layer on the epidermis contained plasmocoagulae, bacterial colonies. Stratum granulosum absent. Acanthosis of the epidermis; distal parts of the spines rounded, in various places connected to each other. Mitoses are present in the nuclei of the cells of the basal layer. Capillaries dilated, pericapillary lymphohistiocytic infiltration with the presence of some eosinophilic leucocytes. Conclusion The histological picture is consistent with psoriasis.

Conclusion/discussion: Although the patient's history and clinical picture, as well as the allergy tests, are consistent with a diagnosis of allergic contact dermatitis, it would probably be useful to perform more extensive investigations to check for the presence of other possible diseases or co-existence of several skin conditions.

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100519 | Angioedema as a first manifestation of urticarial vasculitis: Single center experience

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Background: Complement component analysis is valuable for differentiating the various types of angioedema. Complement C4 may be decreased in systemic lupus erythematosus (SLE), glomerulonephritis, immune complex disease, cryoglobulinemia, hereditary angioedema (HAE), and congenital C4 deficiency. All of the listed conditions can be associated with angioedema.

Method: We describe seven female patients (mean age at the beginning of the disease 36.2 ± 10.8 years) who presented with non-pitting swelling of the face, lips and/or eyelids.

Results: Since angioedema did not respond to antihistamines, HAE was suspected. In 3/7 patients angioedema was accompanied by urticarial and/or purpuric lesions from the beginning. All the patients have significantly decreased levels of C4 in the presence of normal amounts and functional activity of C1 inhibitor (C1-INH). In 4/7 patients decreased levels of C3 were determined. Levels of C1q in sera of 3 patients were found to be slightly reduced or normal. In all of the patients very high concentrations of anti-C1q antibodies were found (in 6/7 levels >100 U/mL). Protein electrophoresis was normal in all patients. Skin biopsy was performed in 3/7 patients. Diagnosis of *hypocomplementemic urticarial vasculitis (HUV)* was confirmed by histology revealing leukocytoclastic vasculitis. Patients were examined regularly over a follow-up period which lasted from 1 to 8 years (mean 4.6±1.5). During that period the first patient developed a full-blown SLE along with a progressive *chronic obstructive pulmonary disease (COPD). Second patient showed the evolution to severe COPD.* The other four patients continued to have only cutaneous manifestations of the disease during the follow-up period of 1 to 4 years. One of them showed only angioedema during the 1 year follow up.

Conclusion: HUV (or anti-C1q vasculitis) is a type III hypersensitivity reaction characterized by urticaria with persistent acquired hypocomplementemia. However, HUV usually includes urticaria like rash, which is not observed with inherited or acquired C1 INH deficiency. The kalikrein-kinin system has been reported to be activated in vasculitis leading to the release of bradykinin. Although HUV is uncommon systemic vasculitis with various clinical manifestations, it is important to be aware that its first presentation may be angioedema.

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DRUG ALLERGY 2

100036 | Natural course of NSAID hypersensitivity: Development of tolerance and progression to chronic urticaria

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Background: Nonsteroidal anti-inflammatory drugs (NSAIDs) are among the most frequently reported agents of drug hypersensitivity, with NSAIDs-induced urticaria/angioedema (NIUA) representing the most common phenotype. NIUA has been suggested to proceed by years the onset of Chronic Urticaria (CU), while a significant proportion of NIUA patients will develop tolerance within six years of the initial diagnosis. Data on the natural course of Single-NSAID-Induced Urticaria/Angioedema and Anaphylaxis (SNIUAA), another clinical phenotype of NSAIDs hypersensitivity reactions, remain scarce. The present study aims to assess the natural course of NIUA and SNIUAA regarding progression rates to CU and the development of tolerance.

Method: We retrospectively evaluated the medical files of patients visiting the Allergy Unit "D. Kalogeromitros" of Attikon University Hospital in Athens, Greece, with a history of acute urticaria induced by NSAIDs. The study comprised three groups a/patients with NIUA (urticaria and/or angioedema to at least two different NSAIDs or positive aspirin Drug Provocation Test) and b/patients with SNIUAA (urticaria and/or angioedema and/or anaphylaxis to a single NSAID of multiple structurally related agents, with tolerance to aspirin or another strong COX-1 inhibitor) and c/ a control group of patients without a history of CU who tolerated NSAIDs. In addition, all patients (groups a and b) were re-evaluated by the end of 2022, 11 ± 9.2 years after the initial reaction, to assess the development of CU and NSAIDs tolerance.