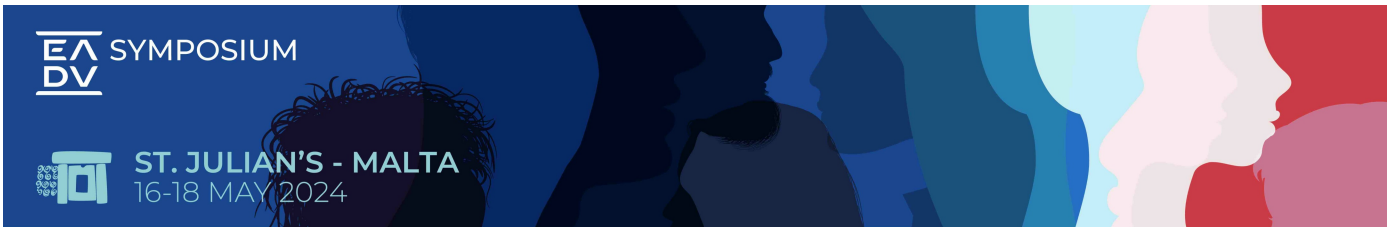

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The paradox of H1-antihistamine hypersensitivity- case report

Alexandra Denisa Oprea*[1], Popa Liliana[1], Chesa Dana[2]

[1]Elias Emergency University Hospital, Dermatology, Bucharest, Romania,[2]Elias Emergency University Hospital, Allergology and Immunology, Bucharest, Romania

Introduction & Objectives: H1** antihistamines (AH) stand as the first-line therapy for a myriad of immunoallergic conditions, including chronic spontaneous urticaria (CSU) and allergic rhinitis. Despite their extensive global usage, hypersensitivity reactions (HR) to AH1 are exceptional events. CSU +/- angioedema are the most commonly reported manifestations of hypersensitivity to AH, displaying a predilection for female adult patients and onset 30 minutes to 6 hours after intake. We describe a case of AH1 hypersensitivity and provide an overview of the potential mechanisms underlying allergies to AH drugs.

Materials & Methods: A 37-year-old man was referred to our clinic for CSU with frequent episodes of exacerbation accompanied by angioedema. The patient had been diagnosed with allergic rhinoconjunctivitis and asthma, for which he was undergoing chronic treatment with beclomethasone dipropionate/formoterol fumarate 100/6 yg inhaler and mometasone nasal spray. The onset of CSU had taken place two years previously following the prophylactic administration of bilastine prior to the first dose of SARS COV2 vaccine. The urticarial lesions subsided spontaneously. The patient was afterwards recommended various AH1 drugs for the control of allergic rhinitis signs and symptoms and each administration induced urticaria flares, facial angioedema and the exacerbation of respiratory symptoms that required multiple courses of corticotherapy. Upon presentation, the suspicion of AH1 hypersensitivity was raised and later confirmed by a positive oral challenge to desloratadine and positive results of basophil activation tests to cetirizine, levocetirizine, desloratadine. The etiologic investigations for CSU consisted in screening for infectious and autoimmune diseases and only revealed the presence of autoimmune thyroiditis. The total serum IgE level was 1038 IU/ml. Corticotherapy was reinstituted with rapid clinical improvement and was followed by the initiation of omalizumab treatment, which was well tolerated and successfully controlled both cutaneous and respiratory allergies.

Results: A high level of suspicion is necessary for the diagnosis of AH1 hypersensitivity. Challenge testing using different drug formulations might be helpful because hypersensitivity drug reactions may not occur in response to the pharmacologically active molecules, but rather to inactive ingredients, including food proteins (gelatin, lactose, starch, cinnamon, cocoa butter). AH1 works as inverse agonists by shifting the H1 receptor to its inactive form. Some authors suggest a paradoxical effect where AH1 may activate the H1-histamine receptor due to its ethylamine group, resembling histamine's molecular composition, causing HR. These observations support the hypothesis that the mechanism in these cases is likely IgE-independent.

Conclusion: Dermatologists should not discard AH1 as potential triggers or exacerbating factors of CSU, if suggested by the patient's medical history. Therefore,** an allergy workup is crucial to avoid missing a potentially life-threatening diagnosis. Omalizumab may be an excellent therapeutic option for achieving disease control in cases of CSU with intolerance to different AH1 treatment regimens.