International Journal of Clinical & Medical Allergy (IJCMA)
ISSN 2332-2799

Barrier Integrity Damage-Elicited Allergic Response (BIDEAR) Syndrome: A Proper Entity?

Case Report

Ervin Ç. Mingomataj[1*], Alketa Bakiri[2,3]

[1] "Mother Theresa" School of Medicine, Dept. of Allergology & Clinical Immunology, Tirana, Albania.
[2] American Hospital of Tirana, Outpatients Service, Tirana, Albania.
[3] Albanian University, Faculty of Medical Sciences, Tirana, Albania.

Abstract

This brief report shows different cases of allergic sensitization elicited by mechanical trauma. The role of the accidental trigger is considered as part of a proper pathological entity, as long as the barrier damage in concert with the pro-allergic immune response was a key factor in the eliciting of the allergic carrier. In this context, we attempted to integrate the event of trauma-related barrier destruction as a constitutive part of a proper diagnostic entity when allergic sensitization is elicited as a result of the mentioned accident, suggesting the term: "Barrier Integrity Damage-Elicited Allergic Response (BIDEAR) Syndrome".

Keywords: Allergic Sensitization; Barrier Damage; Pro-Allergic Immune Response.

Introduction

The disturbed permeability barrier plays an important role in numerous inflammatory diseases [1]. Among them, allergic sensitization through the skin or other barriers occurs when barrier function is disrupted by genetic predisposition, mechanical damage, or the enzymatic activity of allergens [1, 2]. This work shows cases of allergic sensitization that are elicited by mechanical trauma, proposing to comprise this decisive event in the nomenclature of pathologic entity.

Cases Presentation

Case 1: A young adult woman showing any allergic symptom is addressed to an otolaryngologist for a plastic intervention on the inner nose. Afterward, she experienced for the first time clinical symptoms of allergic rhinitis, and actually, she is under appropriate pharmacological treatment.

Case 2: A preschool-aged boy, diagnosed with IgE-mediated egg allergy, reported the development of recurrent urticarial reactions immediately after consumption of eggs-containing foods since early infancy. For the first time, these symptoms occurred few weeks after treatment of partial skin combustion with a traditional mixture containing over-boiled and baked eggs yellow. Gradually, the allergic symptoms are resolved spontaneously during his adolescence.

Case 3: A young adult woman wearing recently ear piercing and earrings, experienced allergic facial skin symptoms few weeks after the beginning of dermabrasion treatment to remove the outermost layers of dead skin cells from the epidermis. She was diagnosed with contact dermatitis to nickel due to a patch test.

Discussion

These case-reports emphasize the decisive role of traumatic damage of epithelial barrier on the development of different allergic diseases due to penetration of allergens in to the deep layers of the living epidermis and increasing of antigen uptake [1, 3]. The underlying barrier defect in concert with pro-allergic immune response (s) are key factors on the eliciting of an allergic carrier in

***Corresponding Author**:
Ervin Ç. Mingomataj,
"Mother Theresa" School of Medicine, Dept. of Allergology & Clinical Immunology, Tirana, Albania.
Tel/Fax: +35542349203
Email Id: allergology@gmx.de

Received: April 09, 2021
Accepted: May 18, 2021
Published: May 24, 2021

Citation: Ervin Ç. Mingomataj, Alketa Bakiri. Barrier Integrity Damage-Elicited Allergic Response (BIDEAR) Syndrome: A Proper Entity?. *Int J Clin Med Allergy.* 2021;06(01):71-72. doi: http://dx.doi.org/10.19070/2332-2799-2100013

affected subjects [1, 4]. Thus, the impaired epithelial integrity during food allergy and histological alterations, indicative of the presence of acanthosis and angiogenesis, are associated with mucosal CD11c+ and CD4+ infiltrates, which suggest the presence of inflammatory immune response to culprit allergen [5]. Moreover, the damaging of the epithelial barrier and Th2 polarization during atopic allergy show modulation of dendritic cell (DC) function, and an innate immune response to allergens with toll-like receptor 9- and toll-like receptor 4-stimulating conjugates [4]. This is possible because the DCs placement at the interface between body surfaces and the environment is ideal for allergen capture, and thereafter, for the inducing of tolerance or initiating and persisting of the immuno-inflammatory response [6, 7].

As long as the function of the DC network is closely controlled and regulated by cytokines released from epithelial cells, the increased allergen uptake due to the absence of regulatory effect after epithelial denudation can induce DCs attraction and activation due to pathogen- and damage-associated molecular patterns [2, 6]. In turn, the DC activation is associated with the up-regulation of co-stimulatory molecules and maturation markers, which enable DCs to activate naïve T cells [7]. In summary, the conceptual framework of epithelial and DC collaboration could explain the process of allergic sensitization after barrier integrity damage [6].

The overriding message distilled from the discussions was that damage to epithelial surfaces lies at the origin of the various manifestations of allergic disease [8]. The epithelium of the nose, skin, etc, which operates as a critical sensor of environmental stimuli under normal conditions, may lead to the development of allergic pathologies when removed by traumatic events. Any diagnostic nomenclatural notion addresses the barrier demolition in the eliciting of allergic pathologies. In these circumstances, we propose to use the term: "Barrier Integrity Damage-Elicited Allergic Response (BIDEAR) Syndrome" to integrate the role of trauma-induced barrier damage in a proper diagnostic entity when the allergic response is triggered immediately after a traumatic accident. The diversity of presented case-reports demonstrates that our concept comprises the affecting of both mucosal and skin barrier by trauma as well as the eliciting of immediate and delayed allergic diseases. Consequently, the analogical term only for the cutaneous pathologies would be "Skin Integrity Damage-Elicited Allergic Response (SIDEAR) Syndrome". Hopefully, the suggested term will find its "citizenship" in the future.

What learns us from this work?

- A damaged epithelial barrier may favor penetration of allergens and develop different allergic diseases;

- The avoidance of epithelial regulation in allergen uptake may lead to persistent inflammatory processes;

- The integration of trauma-related epithelial damage as a part of proper entity suggests us to use the term: "Barrier Integrity Damage-Elicited Allergic Response (BIDEAR) Syndrome".

- This notion can comprise the affecting of mucosal and skin barrier and the eliciting of immediate and delayed allergic pathologies.

References

[1]. Proksch E, Dähnhardt D, S Dähnhardt-Pfeiffer, Fölster-Holst R. Epidermale Barrierestörung bei Dermatosen [Epidermal barrier disorders in dermatoses]. Hautarzt. 2016 Nov;67(11):907-921. German. Pubmed PMID: 27770133.

[2]. Deckers J, Sichien D, Plantinga M, Van Moorleghem J, Vanheerswynghels M, Hoste E, et al. Epicutaneous sensitization to house dust mite allergen requires interferon regulatory factor 4-dependent dermal dendritic cells. J Allergy Clin Immunol. 2017 Nov;140(5):1364-1377.e2. Pubmed PMID: 28189772.

[3]. Filon FL, D'Agostin F, Crosera M, Adami G, Bovenzi M, Maina G. In vitro absorption of metal powders through intact and damaged human skin. Toxicol In Vitro. 2009 Jun;23(4):574-9. Pubmed PMID: 19490843.

[4]. Hosoki K, Boldogh I, Sur S. Innate responses to pollen allergens. Curr Opin Allergy Clin Immunol. 2015 Feb;15(1):79-88. Pubmed PMID: 25546327.

[5]. Rosace D, Gomez-Casado C, Fernandez P, Perez-Gordo M, Dominguez MDC, Vega A, et al. Profilin-mediated food-induced allergic reactions are associated with oral epithelial remodeling. J Allergy Clin Immunol. 2019 Feb;143(2):681-690.e1. Pubmed PMID: 29705246.

[6]. Lambrecht BN, Hammad H. Dendritic cell and epithelial cell interactions at the origin of murine asthma. Ann Am Thorac Soc. 2014 Dec;11 Suppl 5:S236-43. Pubmed PMID: 25525726.

[7]. von Garnier C, Nicod LP. Immunology taught by lung dendritic cells. Swiss Med Wkly. 2009 Apr 4;139(13-14):186-92. Pubmed PMID: 19137454.

[8]. DeKruyff RH, Zhang W, Nadeau KC, Leung DYM, Wills-Karp M. Summary of the Keystone Symposium "Origins of allergic disease: Microbial, epithelial and immune interactions," March 24-27, Tahoe City, California. J Allergy Clin Immunol. 2020 Apr;145(4):1072-1081.e1. Pubmed PMID: 31926182.